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Ill-Timed: The Effect of Early Chronic Illness Onset on Young Adult Psychosocial Development

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ILL-TIMED:

THE EFFECT OF EARLY CHRONIC ILLNESS ONSET ON YOUNG ADULT PSYCHOSOCIAL DEVELOPMENT

by

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Under the Direction of Anthony R. Hatch, PhD

ABSTRACT

Chronic illness affects nearly half of all American adults, yet this experience is often regarded as socially normative for older adults. In this study, I examined chronic illness onset early in the life course and its effects on mastery, a person's self-perception as capable of coping with and managing life's circumstances, and depressive symptoms as informed by the life course perspective and the stress process model. Using multilevel modeling of American Changing Lives Survey (ACLS) data, I examined the following questions: What is the relationship between early onset chronic illness and mastery? Second, what is the relationship between early onset chronic illness and depressive symptoms? Does mastery mediate the relationship between early

onset chronic illness and depressive symptoms? Is early onset chronic illness (24-35) more strongly associated with decreased mastery and increased depressive symptoms than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)? Lastly, does mastery mediate or moderate the relationship between timing of illness onset and depressive symptoms? Through this study, I aim to contribute to sociological knowledge of whether and how chronic illness impacts mastery and depression among young adults. I argue that ill-timed chronic illness impacts young adults' sense of control over their lives, which has enduring psychological and social consequences. Findings support that healthy and chronically ill young adults do not significantly differ on mastery, but ill young adults report significantly higher depressive symptoms than healthy same age peers. Mastery moderates the effects of timing of illness onset on depressive symptoms with older adults reaping greater benefit from mastery against depressive symptoms than young adults with early onset illness. These findings suggest that early onset chronic illness positions people at greater risk for poor mental health outcomes and that the chronic illness experience and its effects are not uniform across the life course. Consequently, work in this area must consider age as an important context in which the life event of chronic illness onset occurs.

INDEX WORDS: Chronic illness, Young adults, Depressive symptoms, Life course, Mental health, Mastery

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DEDICATION

This work is dedicated to the memories of my “Lil Auntie”, Mrs. Jeannie McGee and my “Tee”, Mrs. Marva J. Westbrook, who loved and encouraged me from my first breath until their very lasts. For their examples and presence in my life, I am forever grateful.

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1 INTRODUCTION

Chronic illness is the enduring subjective experience of illness that is initiated by onset of symptoms that are not expected to remit during an individual's lifetime (Bury 1982). Examples of chronic illnesses include asthma, epilepsy, Type 1 diabetes, heart disease, and migraine headache. While some chronic illnesses may onset among younger people, this experience is often normatively linked to the process of aging—older adults (65 and older) expect to experience some form of chronic illness. As a result, individual responses to illness by chronically ill older adults may differ from those of young adults,[1]for whom chronic illness onset is an atypical and unanticipated event (Wickrama et al 2008).

Although much is known about the immediate and short term impact of chronic illness onset during childhood (Harambat 2012, Maslow 2011), adolescence (Siegel et al. 1990), mid-life (Stenholm et al. 2014, Wikman et al. 2011), and late-life (Hughes et al. 2014, Radcliff et al. 2013), less is known about chronic illness onset during young adulthood (Berge et al. 2013). Furthermore, even less is known about the long term consequences of chronic illness onset during this specific phase of the life course.

1.1 Chronic Illness as a Life Event

Chronic Illness is a significant and disruptive life event (Bury 1982, Corbin et al. 1984, Hollinghaus and Utz 2012). A life event is a singular occurrence that initiates a transition from one social status to another (Elder and Giele 2009). Chronic illness onset functions as a significant life event in three primary ways. Specifically, chronic illness onset necessitates engagement in "biographical work" (Corbin and Strauss 1985), is disruptive to the ill individual

[1] Developmental theorists have categorized the ages of 18 to 40 (Erikson and Erikson 1997) and 26-35 (Arnett 2000) as young adulthood. However, in this study's analyses, young adulthood refers to the ages of 24 to 35 years of age exclusively. This construct is discussed in greater detail in Chapter 3 under measurement and sample description.

and their social networks (Lieberman and Fisher 1995), and threatens individuals' social participation (Beatty 2012).

Biographical work, "the continual or occasional reconstruction of one's life" (Corbin and Strauss 1985: 231) occurs through shifts in behaviors, changes in social arrangements, or the development and mobilization of psychological resources. Key sociological research in the area of chronic illness suggests that illness onset significantly disrupts the biography of the affected individual and necessitates personal adaptation (Bury 1982, Williams 1984). For example, using this conceptualization of chronic illness, Peláez-Ballestas et al. (2012) found that Mexican adults (17-66) with Ankylosing Spondylitis[2] reported significant disruption in their identities and thus, sense of belonging to various social networks, including the family.

A number of previous studies have examined chronic illness as a life event within the contexts of mid-life and late-life (Gignac et al. 2000, Lyons et al. 2009).[3] Studies that have examined chronic illness among young adults (Barakat and Wodka 2006, Sparud-Lundin et al. 2010) have regarded chronic illness onset as particularly disruptive to young people's biographies, social networks, and social participation due to the non-normative life stage in which it occurs. In a study of young adults (18-30) with Type 1 diabetes, Sparud-Lundin et al. (2010) found that ill young adults' identities were simultaneously challenged by the developmentally normative processes of redefining interpersonal relationships and establishing independence and the socially disruptive experience of illness. Irrespective of age, however, chronic illness is typically a disruptive life event with wide reaching impact on the individual. Adults with chronic illness report greater functional impairment (Hays et al.1995), more

[2] A chronic inflammatory arthritic condition in which the sacroiliac joints of the hips, pelvis and spine are primarily affected.

[3] Developmental scholars refer to mid-life in multiple ways, such as 40 - 64 years of age (Levinson 1986) and 35-65 (Erikson and Erikson 1997).

stagnated career trajectories (Stroup et al. 2001), and poorer self-esteem (Simoni 2006) than adults without chronic illness. Further discussion of chronic illness as a life event is presented in Chapter 2 (literature review).

1.2 Chronic Illness and Depressive Symptoms

Chronic illness is also disruptive to people's mental health. Specifically, onset of a chronic illness positions a person at increased risk for mental distress, particularly depressive symptoms (e.g. Kanner and Palac 2000, Liew et al. 2011, Turner and Noh 1988). Previous studies have examined if the chronic illness-depressive symptoms relationship differs by specific diagnosis (Macdonald 1988), comorbidity (Anderson et al. 2001, Egede 2005), and impairment (Brown and Turner 2012, Ormel et al. 1997, Turner and Wood 1985). In a study of adults diagnosed with diabetes, Egede (2005) found that odds of diagnosis with a depressive disorder significantly increased with each additional comorbid condition and that levels of depressive symptoms depended on diagnosis, with heart disease and arthritis yielding the greatest odds for depressive symptoms. Similarly, in a study of older adult women diagnosed with multiple subtypes of arthritis, Mingo et al. (2008) found that those who reported more symptomatic and thus, painful arthritis also reported more depressive symptoms. Findings like these suggest that chronic illness' association with depressive symptoms is informed by characteristics of the illness and that chronic illness may not be a uniform event with a singular outcome.

These findings are mirrored in studies that have examined the chronic illness – depressive symptoms relationship among the young (Siegel et al. 1990, Turner and Noh 1988). In their pivotal study examining the relationship between chronic illness, depressive symptoms, and self-esteem among adolescents (12-18), Siegel et al. (1990) found that adolescents diagnosed with sickle-cell anemia, diabetes, and asthma reported significantly higher levels of depressive

symptoms than did healthy peers. Similarly, Turner and Noh (1988) compared the depression trajectories of physically disabled young adults (18-44), middle age adults (45-64), and older adults (65+) to depression trajectories of healthy same-age peers, and found that within each age group, those with disabilities experienced increased risk of depression. Findings like these suggest that the chronic illness - depressive symptoms relationship exists across the life course.

1.3 Chronic Illness and Mastery

Chronic illness as a predictor of depressive symptoms is established and widely agreed upon among scholars (Liew et al. 2011, Macdonald 1988), consequently, the multiple mechanisms by which this relationship occurs have also been identified. The primary psychological mechanism by which chronic illness has an effect on depressive symptoms is mastery (Turner and Lloyd 1999), one's self-perception as capable of coping with and managing life's circumstances (Turner and Noh 1988). Chronic illness is disruptive and harmful to a person's sense of mastery (Pudrovska 2010). Features of some chronic illnesses, such as increased impairment and disability, are associated with decreased mastery (Graff et al. 2009, Turner and Wood 1985), as limitations influence a person's sense of control over life circumstances. Among adults diagnosed with Inflammatory Bowel Disease (IBD)[4], Graff et al. (2009) found that patients who reported higher levels of impairment due to painful symptoms also reported significantly lower levels of mastery than did healthy adults or others diagnosed with IBD, but asymptomatic. Thus, a person's mastery is linked to their health status and characteristics of one's illness informs self-reported mastery. An extensive review of mastery and its buffering effects against disruptive life events and their undesirable outcomes is provided in Chapter 2.

[4] A body of chronic diseases of the gastrointestinal system, including Ulcerative Colitis and Crohn's Disease in which the digestive tract cyclically becomes inflamed and progressively damaged.

1.4 **Chronic Illness and Age**

In addition to being a disruptive life event that increases risk for depressive symptoms (Gunn et al. 2012) and contributes to decreases in mastery (Cott et al. 1999), chronic illness is a life event that occurs at every period of the life course. However, prevalence differs significantly by age group. In the general population, chronic illnesses among children are uncommon. Estimates of prevalence of pediatric cases of non-life threatening chronic illness range from 8 percent (NCHS 2007) to 23 percent (Anderson and Horvath 2004) of children. Among American children, the most common conditions are eye conditions, asthma, and other respiratory disorders (Anderson and Horvath 2004, Torpy 2010).Among this age group, the life areas most directly affected by illness onset are family and peer relationships and formal education (Maslow et al. 2011).

Chronic illness is common among American adolescents (NIH 2013); however, onset of illness during this period is rare[5,6] (Mackner and Crandall 2006).When illness onset does occur, the primary life areas impacted are peer and family relationships (La Greca et al. 1995) and formal education. Scholars (Emerson et al. 2009, Erkolahti and Ilonen 2005) debate if chronically ill adolescents experience more deficits in academic performance, school attendance, and extracurricular activity participation than healthy peers. Yet, when challenges do occur during adolescence, these early deficits remain influential in young adulthood and contribute to disparities in employment, education, and wealth between the chronically ill and healthy (Maslow 2012).

[5] About 1 in 4 American adolescents have a chronic illness, however, many of these cases reflect congenital and childhood onset.

[6] Irritable Bowel Disorder and Type 1 diabetes are the most prevalent chronic health conditions that initially present during adolescence (12-19).

Chronic illness is prevalent among young adults. According to the Center for Disease Control (2009), approximately 1 in 5 American young adults[7] report a chronic health condition, with asthma, diabetes, arthritis and hypertension as the most common conditions among this age group. When illness onset occurs during this period in the life course, young adults often involuntarily withdraw from education, vocational training, and the workforce due to functional impairment and inability to meet expectations of consistent attendance (Boot et al. 2010) and performance (Bevan et al.2013). This withdrawal from the practical preparation and performance of the role of worker is significant because young adulthood is socially regarded as the primary stage for securing financial independence needed for entry into full adulthood (Serido and Shim 2014, Xiao et al. 2014).

As illustrated above, even among the young, chronic illness becomes increasingly more common with each progressive life stage. This pattern continues into the later stages of the life course. In fact, by mid-life, nearly 1 in 3 American adults reports a chronic illness and by late-life, 1 in 2 older adults report a chronic illness (Paez et al. 2009). These age related differences in prevalence of chronic illness and the social perceptions of normativity that these differences might confer introduce interesting questions about how age or more specifically, timing of onset structures chronic illness as a disruptive life event. Since most Americans who become chronically ill do so at mid-life or late-life (Ornstein et al. 2013, Paez et al. 2009), chronic illness prior to one of these stages is less typical, earlier than expected, and thus, non-normative.

In the section below, I briefly introduce two competing explanations for how timing of chronic illness onset might frame the relationships between 1) chronic illness and mastery and 2) chronic illness and depressive symptoms. These explanations are referred to henceforth as the cumulative

[7] In this CDC report, "young adults" refers to adults between the ages of 18 and 30 years old.

disadvantage and youthful resilience explanations and are more thoroughly discussed in Chapter 2.

1.5 Cumulative Disadvantage versus Youthful Resilience

Two competing explanations organize this study, cumulative disadvantage and youthful resilience. The cumulative disadvantage explanation derives from cumulative disadvantage theory (Ferraro and Moore 2003) that posits that early advantage or disadvantage situate a person for continued advantage or disadvantage at later stages of the life course. As a disruptive life event that decreases mastery (Turner and Wood 1985) and increases depressive symptoms (Simoni et al. 2006), chronic illness positions all people for more disadvantage in some life areas (e.g. physical functioning) when compared to healthy people. However, the cumulative disadvantage explanation offers a way of comparing the relative disadvantage experienced by subsets of chronically ill people who become ill at different life stages. According to cumulative disadvantage theory (Ferraro and Moore 2003), a disadvantage experienced early situates a person for continued disadvantage at later stages of the life course (Dannefer 2003, Diprete 2005). Consequently, early onset chronic illness may situate younger people for more disadvantage (e.g. less mastery, greater depressive symptoms) than people who experience chronic illness onset later during more socially normative times.

Alternatively, the youthful resilience explanation derives from a body of resilience research (Karoly and Ruehlman 2006, Norris et al. 2009) that suggests that immature cognitive and neuropsychological development inherent to youth act as protective buffers against disruptive events and their long term consequences (Uswatte and Taub 2009). Resilience is "the ability of an individual to function competently in the face of adversity or stress" (Murphey et al. 2013). Although older adults frequently self-report better well-being and lower depressive

symptoms in the face of disruptive life events than do younger people (Chapman and Perry 2008, Fisk 2009), children, adolescents and young adults receive more psychological protection from cognitive and emotional "plasticity" (Easterbrooks et al. 2013), which can contribute to resilience when faced with stressors (Davidson and McEwen 2012, Karatsoreos and McEwen 2011). Plasticity refers to the idea that the developing brain in early life is more flexible and adaptable to endogenous and exogenous change than more mature brains (Stiles 2000). This is a common argument within developmental psychological and neuropsychological literature (Davidson and McEwen 2012), however, some scholars (e.g. Stiles 2000, Utwatte and Taub 2012) contend that the brain remains pliable into late-life, but to a lesser degree than infancy through young adulthood.

The cumulative disadvantage and youthful resilience explanations provide two alternative justifications for why timing of illness onset might be important in understanding this disruptive life event's effects on a person's mastery and experience of depressive symptoms. This project is titled "Ill-timed" because when in life a disruptive event (Depreter et al. 2013, Tavernier and Willoughby 2012) occurs provides context for the meaning and consequences of that event (Burton 1996, Moen 2001). While the cumulative disadvantage explanation asserts that early onset illness positions a person to accumulate more disadvantage across the life course than later onset; the youthful resilience explanation suggests that youth may protect people from the harmful effects of early onset chronic illness. Consequently, the cumulative disadvantage and youthful resilience explanations provide two different ways of thinking about if and how timing of chronic illness onset differentially positions those with early onset chronic illness for better or worse coping and mental health than healthy same-age peers and chronically ill people with mid-life and late life onset. Below, I present the aims of this study, the research questions that guide

this study, a brief overview of the methods employed in this study, and the organization of the remainder of the document.

The aims of this project are 1) to apply a life course perspective to the examination of chronic illness onset, 2) to situate early and thus, "ill-timed" chronic illness within the stress process model as a primary stressor, an initial disruptive event or experience that begets secondary stressors, additional disruptive events and strains and 3) to explore the process through which mastery mediates and/or moderates the mental health effects of chronic illness onset. The sociological significance of this particular project is found in its attention to an understudied population, chronically ill young adults, and its distinct experience with chronic illness. In order to meet these aims, I examined the following research questions in this study:

1. What is the relationship between early onset chronic illness and mastery? [8]

2. What is the relationship between early onset chronic illness and depressive symptoms? Does mastery mediate the relationship between early onset chronic illness and depressive symptoms?

3. Is early onset chronic illness (24-35) associated with lower mastery than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)?

4. Is early onset chronic illness (24-35) associated with greater depressive symptoms than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)? Does mastery mediate or moderate this relationship?

To answer these research questions, I conducted analyses using secondary data from the American Changing Lives study. In these analyses, I performed multilevel modeling of panel data collected from American adults age 24-96 years old. Using waves 1-4 of data from this study, I modeled random intercepts and random slopes to examine the relationships between timing of chronic illness onset and 1) mastery and 2) depressive symptoms. I provide a full

[8] Early onset refers to onset that occurs prior to mid-life and late-life, which in this study includes the ages of 36 -64 and 65 and older, respectively.

description of the research methods employed in this study in chapter 3. Analyses and results pertaining to the effects of chronic illness onset on mastery are presented in chapter 4. Chapter 5 presents analyses and results pertaining to the effects of chronic illness onset on depressive symptoms. Lastly, in the concluding chapter 6, I review findings in relation to the cumulative disadvantage and youthful resilience explanations, discuss the sociological significance of results and direction for future scholarship in the areas of chronic illness and life course studies.

1.6 Conclusion

In conclusion, chronic illness is an area ripe for sociological study, particularly as it relates to psychological coping and mental health. As a disruptive life event that half of all Americans can expect to experience (CDC 2009, Paez 2009), chronic illness is a common social phenomenon with deeply personal costs and broader social and economic implications. Through this project, I make three primary contributions to the field, 1) focused attention to the chronic illness-mastery association among adults with early onset chronic illness, 2) examination of early onset chronic illness as a stressor that indirectly contributes to depressive symptoms, and 3) the within group comparison of age cohort differences in changes in coping resources and mental health outcomes among the chronically ill. Moreover, the study's analyses of longitudinal data are a particular strength, as no identified work has examined the effects of chronic illness onset on trajectories of mastery and depressive symptoms across stages of the adult life course. Thus, this project aims to make both theoretical and methodological contributions to health and life course research on the effects of chronic illness onset.

In the chapter that follows, I review literature most pertinent to the study of timing of chronic illness onset, mastery, and depressive symptoms in addition to discussing the theoretical frameworks in which this study is embedded.

2 LITERATURE REVIEW

In this chapter, I review literature on the life course perspective (Elder 1994), the stress process (Pearlin and Schooler 1978), and depressive symptoms (Turner et al. 1999). This literature review is followed by an elaborated discussion of the cumulative disadvantage and youthful resilience explanations that I introduced in Chapter 1. This discussion is followed by a critique of the literature and the hypotheses that were developed in response to gaps identified in the literature. Lastly, the chapter concludes with a brief overview of the organization of the remainder of this study.

2.1 Life Course Perspective

The life course perspective provides a theoretical paradigm through which to investigate transitions, trajectories, and events in the life of an individual or cohort (Elder 1985). This framework highlights the significance of historical context, timing in lives, interpersonal relationships and personal agency in understanding the meaning of lived experiences (Elder 1994). The life course is socially structured by collectively held expectations of normative social roles and statuses for each life stage (Neugarten 1965, Elder 1994). Fundamentally, the life course perspective conceptualizes the life course as "a lifelong manifold of intertwining cumulative processes, in which earlier events and experiences are consequential for later events and experiences and their management by individuals" (Elder and Giele 2009: 123-124). This perspective is central to this study, which examines how a single, yet significant life event, chronic illness onset, influences not only the development of individual psychosocial resources, but mental health across adulthood (Elder et al.1996, Hennighausen et al. 2004). Although prior research has applied a life course perspective to examining chronic illness during childhood and adolescence (Maslow et al. 2011, Maslow 2012), health researchers have yet to examine chronic

illness during young adulthood as a predictor of mastery or depressive symptoms at later stages in the life course.

2.1.1 The Life Event

A life event is a singular occurrence that initiates a transition from one social status to another (Elder and Giele 2009). This transition often necessitates the reconfiguration of social networks, renegotiation of self-concept, and reliance on coping resources (Bury 1982). Chronic illness onset is a significant life event that initiates the transition from the social status of healthy to that of ill. Previous scholarship has acknowledged that this transition necessitates changes in interpersonal relationships (Paleaz-Ballestas et al. 2012, Williams 1984), disrupts individuals' personal biographies and self-concepts (Bury 1982), and threatens self-esteem and personal mastery (Turner and Butler 2003). Other disruptive life events that have received attention in the literature include divorce (Shek 2007), widowhood (Hahn et al. 2014), and involuntary unemployment (Moen 2001).

Although much of the sociological literature has focused on disruptive life events, the transition into socially desirable statuses, such as being married (Wickrama et al 2013) and becoming a parent (Umberson et al. 2011), has also received attention in the literature. A life event is not only characterized by its potential to cause harm or disrupt, but also by the timing of its occurrence, and its short and long-term consequences (Depreter et al. 2013, Tavernier and Willoughby 2012). A life course perspective highlights the importance of when in the life course an event occurs. Of principal importance is the life course concept of timing in lives, in which the definition and significance of an event depend on when in one's life it occurs (Elder and Giele 2009). This useful theoretical concept has been applied to studies examining various early and thus, "off time" transitions, including adolescent parenthood (Burton 1996), retirement from

paid work (Moen 2001), and adolescent transition into adult roles of employee and caregiver (Hagan and Wheaton 2003).

The functional impairment (Egede 2005), financial obligations (Kahn and Pearlin 2006), and psychological stress (Wiebe et al. 2005) associated with chronic illness onset likely vary depending on age at illness onset. For example, Wiebe et al. (2005) examined how the stressor of illness onset necessitates mobilization of complex coping strategies, like cognitive restructuring, that are beyond the emotional and developmental capacity of chronically ill children and adolescents. As a result, chronically ill children and adolescents' ability to cope with the psychological stress that accompanies illness depends most directly on caregivers' abilities to model effective mobilization of coping resources, namely problem solving (Comeaux and Jaser 2010). Alternatively, when chronic illness onset occurs during young adulthood, studies (Saunders et al. 2011) suggest that social stressors in peer relationships are particularly salient. In a study of social drinking among young adults with Type 1 diabetes and Inflammatory Bowel Disease, Saunders et al. (2011) identified how internal and external social pressures to engage in age dependent socially normative behaviors, like frequent alcohol consumption, resulted in ill young adults ignoring negative health consequences in favor of perceived normalcy.

As detailed in these studies, age or timing in lives is essential in defining the experience and effects of chronic illness onset across the life course. Life events are characterized as occurring early, on-time, or late (Burton 1996). There has been considerable study of how timing of life events gives meaning to the event and influences outcomes for the individual. Much of this work has examined transitions into and out of social roles, including grandparent (Burton 1996), retiree (Verrill 2002), and spouse (Carlson 2012). The characterization of an event as occurring early or on-time is dependent on the nature of the event, social context in which it

occurs, and the ways people ascribe meaning to the event and the individual who experiences it (Elder 1994, Hutchinson 2010). For example, Carlson (2012) found that the evaluation and psychological impact of the transition to marriage was conditioned by personal expectations regarding desired and thus, normative timing of the event. Adults who experienced the transition "off-time" at undesirable ages experienced significantly more psychological distress than those who experienced marriage "on-time." Like marriage, participation in other social institutions, such as paid employment and formal education is perceived as developmentally normative for young adults (Gitelson and McDermott 2006). For young people who experience early onset illness, participation in these normative tasks are frequently disrupted by illness onset (Driedger 2003, Fuligni and Pederson 2002). Alternatively, when chronic illness presents during late-life, social expectations of productivity in education and paid work are minimal (Kahn and Pearlin 2006). Thus, timing of a life event's occurrence frames the social meaning and consequence of that event.

In conclusion, a life course perspective provides a framework for examining chronic illness onset as a life event that initiates a transition in status and influences a person's psychological coping resources and mental health across subsequent life stages (Elder et al. 1996). In conjunction with a life course perspective, the stress process is useful for explaining if and how the disruption of chronic illness onset is patterned by when in life illness onset occurs (Driedger 2003, Fuligni and Pederson 2002). In the section that follows, I review the stress process and focus on disruptive life events as stressors, mastery as a mediating and moderating factor, and this theory's prior application to the study of chronic illness.

2.2 The Stress Process

The stress process model provides a framework for examining interconnections between the causes of stress, the mediators of its effects, and its psychological, physical, and social outcomes (Pearlin et al. 1981: 337). One of the useful features of this model for understanding the onset and experience of chronic illness is the way in which micro and macro-level processes are thought to work together to shape the impact of social stress on the life course (Pearlin et al. 1981). Specifically, this model outlines how social stratification directly and indirectly contributes to unequal exposure to disruptive life events, chronic life strains, and social statuses between individuals and groups (Pearlin 1989). Social statuses that have received considerable attention in the literature include race (Miller et al. 1995, Oates and Goode 2013), educational attainment (Pearlin and Schooler 1978, Schieman et al. 2003), and sex (Falci 2011, Nolen-Hoeksema et al. 1999, Thoits 1987).

Additionally, the stress process model (figure 2.1) highlights the mediating role of psychological and social resources in the relationship between stressors and their multiple effects across various life domains (Pearlin et al. 1981). The stressor of chronic illness onset significantly and simultaneously impacts life domains, such as identity (Dickson et al. 2008), family life (Chen and Fish 2013), work (Beatty 2012), and general social participation (Reissman 1990). Thus, the effects of chronic illness onset on these and other life domains may be mediated by the psychological resource, mastery (Pearlin et al. 2007). Moreover, the stress process is useful for examining how variations in this resource may explain the development of depressive symptoms.

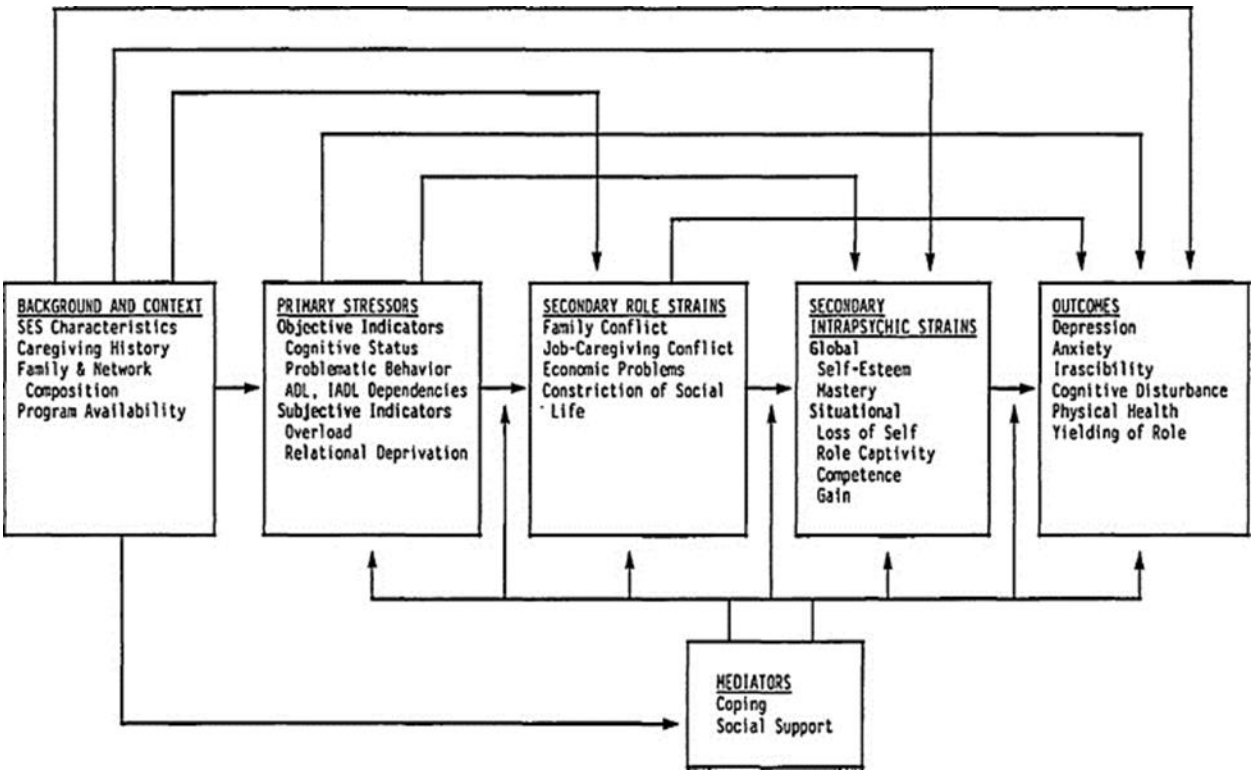

Figure 2.1 The Stress Process Model (Pearlin et al. 1989)

As a stressful life event, chronic illness onset may disrupt normative psychosocial development by eroding one's sense of mastery, which may increase depressive symptoms (Mingo et al. 2008, Turner et al. 1999). Due to the involvement of numerous microlevel processes, the stress process model is especially applicable to the study of chronic illness onset and its effects on psychosocial resources and mental health. In the section below, I discuss mastery across the life course with particular attention to mastery's function as mediator within the stress process.

2.2.1 Mastery

In review of the stress literature, mastery acts as a particularly influential mediator between diverse stressors and outcomes (Avison and Cairney 2003, Mirowsky and Ross 2003, Pudrovska et al. 2005, Yang 2006). Mastery has been found to serve a protective function against the negatives effects of numerous stressors, including chronic illness onset (Dickson et al. 2008),

racial discrimination (Clark et al. 2002, Watkins et al. 2011), and socioeconomic disadvantage (Kiviruusu et al. 2013, Mirowsky and Ross 2011). In the general population, mastery is positively associated with self-esteem (Simoni et al. 2006) and better self-reported health (Cott et al. 1999). Generally, mastery has a curvilinear shape over the life course, with increases from childhood through middle adulthood and decreases in late-life (Turner and Schieman 2008). Mastery is acknowledged as the most important psychological resource in protecting a person from the psychologically damaging impact of undesirable life events (Mirowsky and Ross 2003) or socially devalued social statuses (Pearlin et al. 1981).

Mastery among children has been studied widely in the fields of education (Geary et al. 2007), developmental psychology (Aunola et al. 2013), and sociology (Carlson and Corcoran 2001). Disruptive events occur throughout the life course, however, when in life these events occur is centrally important in understanding the event's immediate and long-term effects on the individual (Hutchinson 2010). A disruptive childhood event that has received significant attention in the literature is chronic illness onset. Most of this work has attended to the experience of children diagnosed with immediately life threatening conditions, like cancer, (Langeveld et al. 2003, Meeske et al. 2001). Children diagnosed with life-threatening conditions report lower mastery than healthy counterparts (Parry 2003). This evidence suggests that even among the youngest, mastery is negatively associated with having a physical health condition.

The protective function of personal mastery is widely affirmed by scholars (Pearlin et al. 1981, Pudrovska et al. 2005, Schieman and Meersman 2004). Much of this literature has focused on mastery during the developmental period of adolescence (Conger et al. 1999). It has been argued that a stronger sense of mastery during adolescence is a determinant of better social, psychological, and physical health outcomes during adulthood (Repetti et al. 2002, Surjadi et al.

2011). For example, in their study of parental influences on adolescent mastery, Surjadi et al. (2011) found that greater parental support at adolescence was associated with greater mastery at adolescence, which predicted higher levels of mastery during young adulthood. Similarly, Conger et al. (1999) found that parental socioeconomic status is predictive of adolescents' mastery, which is related to more effective adolescent problem solving. Mastery is essential to successful adolescent development because the belief that one can effectively cope with varied circumstances, such as undesirable events or unexpected change, is necessary for complex problem solving and bolstering of self-concept as competent (Kroger 2000). Thus, mastery is a necessary tool at and beyond adolescence for the management of the developmentally normative tasks of maturation and assumption of more complex social roles.

As with children and adolescents, mastery among young adults is an essential psychological resource for managing disruptive life events (Aneshensel 1992, Pearlin et al. 1981). However, young adults report considerably lower mastery than adults at mid-life (Mirowsky and Ross 2003), suggesting that this resource and the psychosocial protection it provides increase with age. When a disruptive life event occurs during young adulthood, less mastery is available to buffer the effects of this enduring stressor (Shanahan and Bauer 2004). The protection conferred by mastery continues throughout the latter stages of the life course, mid-life and late-life. Mastery continues to increase through mid-life until plateauing and subsequently, decreasing during the latter stages of late-life (Miller et al. 1995, Pearlin et al. 2007). Among middle age and older adults, mastery has been found to buffer the effects of chronic stressors other than chronic illness, such as racial discrimination (Watkins et al. 2011). While examining the effects of discrimination on the development of depressive symptoms among African American men, Watkins et al. (2011) found that across age groups, a greater

sense of mastery most strongly and significantly protected men from the most harmful psychological effects of discrimination. Moreover, this study illustrated the importance of age in determining the effect of stressors, as the discrimination-depressive symptoms relationship was strongest for men between 35-54 years old.

As with other valuable resources, studies suggest that racial, socioeconomic, and gender disparities in personal mastery exists, with white adults, individuals with higher socioeconomic status, and men reporting greater mastery on average than people of color, individuals with lower socioeconomic status, and women (Mirowsky and Ross 2003, Mirowsky and Ross 2007). Differences in mastery by health status have also received widespread attention in the sociological literature (Cott et al. 1999, Dickson et al. 2008). Mastery has been found to buffer the effects of increasing functional impairment (Yang 2006), chronic pain (Pudrovska et al. 2011), and general uncertainty related to managing a chronic health condition (Charmaz 1995). Those with greater mastery weather the challenges of illness better than those who perceive themselves as more limited in their capacity to cope (Pudrovska 2005). For example, among middle age people diagnosed with Chronic Fatigue Syndrome (CFS)[9] Dickson et al. (2008) found that illness onset primarily impacted individuals' self-concepts through their loss of a sense of mastery. Dickson et al. (2008) found that individual differences in chronically ill adults' identity loss, negotiation, coping, and eventual acceptance of diagnosis were primarily explained by individual variations in mastery. The majority of literature on the effects of chronic illness onset on mastery has focused on middle age (Dickson et al. 2008) and older adults (Pudrovska et al. 2005, Yang 2006). These studies have highlighted the protective function of mastery against

[9] A chronic disease of unknown origin characterized by long lasting physical fatigue, cognitive dysfunction and sleep disturbance.

depressive symptoms (Turner and Butler 2013), erosion of self-esteem (Turner and Noh 1988), and increased mortality (Surtees et al. 2006).

As illustrated above, scholars have consistently demonstrated the significance of mastery as a mediating factor between stressors and outcomes, particularly using samples of middle age and older adults (Jonkker et al. 2008, Pearlin et al. 2007). For example, Yang (2006) highlighted mastery's mediating role in the relationship between functional impairment and depressive symptoms among older adults. Yang (2006) found that disabled older adults who reported a greater sense of mastery at Wave I reported lower levels of depressive symptoms on the Center for Epidemiologic Depression Scale (CESD) at Wave II than did those who reported lower levels of mastery at Wave I.

Even among studies examining mastery's mediating effects within samples including young people, (McQuillan et al. 2003, Pudrovska et al. 2005, Simoni et al. 2006), age at onset has not received adequate attention. In a study of indigent women living with HIV, Simoni et al. (2006) noted that mastery mediated the relationships between quantity and quality of social support and depressive symptoms. However, discussion regarding if and how the social support-depressive symptoms relationship may differ depending on when in a woman's life she is diagnosed is negated. One notable exception is Ruelhman et al.'s (2010) study of the correlation between psychosocial resources, including mastery, and chronic pain and illness among young adults. Ruelhman et al. (2010) found that young adults (17-24) diagnosed with depression reported significantly lower levels of mastery than healthy peers. Other exceptions include Taylor and Turner's (2002) study of the effects of discrimination on depressive symptoms during late adolescence and young adulthood and Turner and Butler's (2003) study of the effects of early life trauma on depressive symptoms at young adulthood. Turner and Butler (2003) found

that coping resources, like mastery and self-esteem, buffered the negative effects of early life trauma and explained differences in depressive disorders during adolescence and depressive symptoms during adulthood. These findings provide evidence for mastery's role as a mediating mechanism through which a stressor, like chronic illness, has an impact on a person's mental health.

As discussed above, the majority of work on mastery in the stress process literature has examined its potential role as a mediator. However, fewer scholars have examined if mastery also *moderates* the effects of stressors on outcomes. One notable exception is Pudrovska et al.'s (2005) study of mastery's dual function as a mediating and moderating influence on the economic hardship-depressive symptom relationship among older adults. Although the authors found support for mastery as mediator, Pudrovska et al. (2005) acknowledged that mastery also conditioned the effects of hardship and depressive symptoms. The positive association was weaker among older adults with greater mastery than among those with lower levels of mastery. Moreover, the authors found that mastery's moderating effects varied by when in the life course the stressor of financial hardship occurred. Consequently, it is reasonable to assume that mastery's moderating properties may extend to another stressful life event, chronic illness onset. Immediately below, I discuss the important stress process concept of stress proliferation and review related work.

According to the stress process, stressors are classified as primary or secondary. This classification refers to temporal order, rather than subjective importance or potency of consequence (Pearlin and Schooler 1978). Primary stressors are first in sequence, while secondary stressors are those that result from the effects of primary stressors and extend into other life domains. For example, a secondary stressor associated with the primary stressor of

chronic illness onset is self-identification as chronically ill. Identification with this socially stigmatized status may produce additional secondary stressors of diminished self-esteem and an eroded sense of mastery. As these resources are essential to effectively coping with the negative consequences of illness onset (Mirowsky and Ross 2003), a person's inability to mobilize them will likely result in additional secondary stressors (Pearlin 1989), such as development of depressive symptoms. This process of stress proliferation occurs when an initial stressor generates multiple additional strains for the affected individual and those within their social networks (Pearlin 1997, Turner and Lloyd 1999).

According to Pearlin (1989: 247-248), stress operates in the lives of individuals through "multiplication and contagion," meaning that initial primary stressors create circumstances favorable to the generation of other stressors that may have an impact beyond the individual. For example, chronic illness catapults people into a stigmatized social category, which may influence their participation and efficacy in macrolevel social structures, such as the workforce (Vickers 2003). For example, Miah and Wilcox-Gok (2007) found that chronically ill workers accumulate fewer assets than healthy peers, and as a result, are significantly less likely to retire early. Similarly, studies support that the chronically ill are less likely to engage in paid labor (Wilson 2001) or receive job promotions (Beatty 2012) when they do participate in the workforce. Viewed in tandem with a life course perspective, findings like those outlined above suggest that chronic illness contributes to disparities beyond physical functioning (Sacco et al. 2013), psychosocial resources (Kotsis et al. 2012), and mental health (Brown and Turner 2012) between the ill and the healthy. Furthermore, these finding offer support for chronic illness as a proliferator of stress and disadvantage.

In the stress proliferation process, the accumulation of disadvantage after illness onset occurs as a result of people experiencing role strain in important life areas, namely employment (Stroup et al. 2001, Vickers 2003). According to Goode (1960: 483), role strain is "the felt difficulty of fulfilling role obligations" experienced by individuals across life domains. For example, in a study of chronically ill employees, Vickers (2003) illustrated how organizational and social expectations of consistency produced role strain for ill employees unable to meet the socially desirable standards of consistent attendance and performance. Per Vickers (2003), the constant threat of negative job performance evaluations, the threat of termination, and preemptive attempts by ill workers to perform in a manner inconsistent with their abilities generated additional stressors. These findings suggest that chronic illness can function as a stress proliferator that contributes to disparities by health status in varied life arenas including self-esteem (Schroevers et al. 2003), wealth (Miah and Wilcox-Gok 2007), and work (Stroup et al. 2001).

As a stressful life event, initiator of chronic strain, and stress proliferator, chronic illness necessitates constant attempts to maintain stability and control (Aujoulat et al. 2008, Gordon et al. 1998), in the face of continuous and inevitable loss and change (Charmaz 1994). Chronic illness onset may decrease young adults' mastery over their lives just at the point at which they are establishing autonomy, cementing identity (Erikson and Erikson 1997), and securing social roles, such as student or employee (Ryder 1965). Thus, the stress process model provides an appropriate framework for exploring if chronic illness onset during this sensitive and developmentally distinct period of the life course explains differences in mastery or depressive symptoms when compared to healthy same aged peers and adults with onset later in life.

In the section below, I review literature germane to the study of depressive symptoms, with a focus on depressive symptoms as an outcome of disruptive life events, prevalence of symptoms across the life course, demographic differences in depressive symptoms, and the chronic illness - depressive symptoms relationship.

2.3 Depressive Symptoms

In the literature, considerable attention has been afforded to depressive symptoms and psychiatric diagnoses among the young (Hood et al. 2006, Pine 1999). Late adolescence through young adulthood is regarded as the most common prodromal stage for persistent and severe psychiatric diagnoses, like schizophrenia and major depressive disorder (Pine 1999). Much of the scholarship in this area has investigated the role of undesirable life events as causal factors in the development of depressive symptoms. Prior studies have examined parental divorce and family restructuring (Langenkamp and Frisco 2008), natural disaster (Warheit et al. 1996), trauma (Frye and Liem 2011), and diagnosis with life threatening diseases (Shroevers et al. 2003) as potential causes of depressive symptoms.

Fewer scholars have examined the relationship between more common and non-life threatening illnesses and depressive symptoms. Studies that have examined this relationship (Hood et al. 2006, Insabella et al. 2007) have limited analyses to adolescents and young adults diagnosed with Type 1 diabetes and relied on cross-sectional data from small predominantly white samples. Findings from these and similar studies (Berge et al. 2013) have established that among young people, chronic illness is positively associated with depressive symptoms.

Although scholars have investigated early onset chronic illness' association with depressive symptoms cross-sectionally, there has been limited study of enduring and cumulative effects across the life span (Frye and Liem 2011, Goodman and Must 2011). However, one

exception in the literature on early onset chronic illness is Hobbie et al.'s (2000) longitudinal study of childhood cancer as a predictor of young adult depressive symptoms, in which the authors highlight early illness as a precipitant of cumulative disadvantage in psycho-emotional development. No identified studies have analyzed longitudinal data to examine the relationship between chronic illness onset during any stage of the early life course and depressive symptoms at mid-life or late-life.

Empirical research is consistent in findings that depressive symptoms decrease across young adulthood and into mid-life and subsequently, increase during late-life (Miech and Shanahan 2000, Mirowsky and Ross 2002). Declines in depressive symptoms during young adulthood occur as people assume more permanent roles and experience relative stability in social placement (Arnett and Taber 1994). This decline continues into early mid-life, which is the point in the life course when depressive symptoms are at the lowest (Mirowsky 1996). Scholars argue that better mental health at mid-life is explained in part by increased likelihood of occupying stable social roles, namely worker, parent, and spousal partner (Kroger and Haslett 1987, Reitzes and Mutran 1994). Moreover, the social benefits of occupying these roles, stable social location and social support, are protective against depressive symptoms (Mirowsky and Ross 1992).

Most studies on depressive symptoms during adulthood have exclusively examined the mental health of older adults (65 years of age and older). These studies have highlighted the harmful effects of disruptive life events, particularly, widowhood (Hahn et al. 2014), retirement (Moen 2001), and declining physical health or ability (Pudrovska et al. 2005). Each of these life events are aptly characterized as losses of socially valued statuses and thus, stressors that are capable of generating additional stressors across life domains and influencing mental health

outcomes (Taylor and Lynch 2004, Turner and Lloyd 1999). In Kessler et al.'s (2010) study of age differences in major depressive disorder occurrence and treatment between older adults (65+) and adults age 18-64, the authors found that the influence of physical health on depressive symptoms weakens with age, suggesting that ill older adults' mental health may be less negatively impacted by their physical health status than younger counterparts. Importantly, this study focused exclusively on age differences in meeting DSM criteria for diagnosis of Major Depressive Disorder and did not account for depressive symptoms that may impact a person's wellbeing, regardless of externally validated severity or diagnosis. These findings suggest that the relationship between physical health and depressive symptoms may be moderated by age and may change across a person's lifetime.

Differences in depressive symptoms between demographic groups, primarily based on race (Kessler et al. 1999), gender (Nolen-Hoeksema et al. 1999), and educational attainment (Schieman and Plickert 2008) have also been studied widely. In the tradition of health disparity research, much of this work has examined if occupying a socially devalued or minority status is associated with poorer mental health outcomes, specifically, greater depressive symptoms (Brown and Turner 2012). For example, Lincoln et al. (2010) examined if changes in depressive symptoms over a 16 year period differed for black Americans and white Americans. This study's findings suggest that patterns of depressive symptoms are heterogeneous within each racial group and these patterns do not significantly differ for white and black Americans. Scholars have also examined gender differences in depressive symptoms and findings support that women report greater depressive symptoms than do men (Falci 2001, Thoits 1987). Lastly, higher education is consistently found to share a negative association with depressive symptoms (Turner et al. 1999) and related mental health disorders, including Major Depressive Disorder (Kessler et

al. 1999). Framed by the stress process, the findings reviewed above suggest that stressors ranging from family reorganization (Frisco and Langenkamp 2008) to occupying a disadvantaged social position (Lincoln et al. 2010, Schieman and Plickert 2008) are associated with increased depressive symptoms among all age groups. In the section below, I focus more directly on literature pertaining to the chronic illness – depressive symptoms relationship.

As evidenced by the literature, the importance of the effects of chronic illness onset on trajectories of depressive symptoms extends beyond increased symptoms or risk of depressive disorders, and extends to physical functioning (Sacco et al. 2013) and increased risk for other mental health conditions (Kim et al. 2000). The mental health outcomes associated with chronic physical illnesses have been widely studied (Beckerman 2011, Kivuruusu et al., 2007, Ruehlman et al., 2010). Empirical and theoretical research supports that chronic illness onset is significantly related to increased depressive symptoms among adults (Bierman et al, 2011, Liew 2011, Schnittker 2005). For example, Schnittker (2005) found that among adults over 50, depressive symptoms are strongly and positively associated with chronic conditions, including arthritis, diabetes, hypertension, and lung conditions. However, the relationship weakens with age, suggesting that an "age-graded effect" is present in the chronic illness-depressive symptoms relationship. Schnittker (2005) and others' (e.g. Turner and Wood 1985) findings suggest that there is a need to examine how this age-graded effect presents across the entire stage of adulthood, including young adulthood.

Scholars have also identified that increased depressive symptoms act as a secondary stressor by diminishing self-perception of functioning and motivation for treatment (Katon 2003). Specifically, depressive symptoms among the chronically ill are negatively associated with chronically ill adults' adherence to medical treatment and self-reported health and

functioning (Ciechanowski et al. 2003, Park et al. 2004). Ciechanowski et al. (2003) found that among adults with type 1 and type 2 diabetes, patients reporting greater depressive symptoms also reported more diabetes symptoms and poorer adherence to recommended exercise and diet regimens. Similarly, among patients with psoriatic and rheumatoid arthritis, Kotsis et al. (2012) identified that patients' levels of depressive symptoms predicted health related quality of life, which was correlated with illness related anxiety, even when controlling for pain and disease severity. These findings articulate the complexity of the chronic illness – depressive symptoms relationship and demonstrate how chronic illness acts as a stress proliferator, creating conditions favorable to the generation of secondary stressors like decreased functioning (Park et al. 2004) and increased risk for additional psychiatric disorders (Kim et al. 2000).

Studies that examine chronic physical illness as a predictor of depressive symptoms have frequently failed to provide insight into *how* chronic illness onset increases depressive symptoms among the chronically ill (Turner and Wood 1985, Turner and Noh 1988). An exception is Beckerman's (2011) study of adults diagnosed with Lupus Erythematous[10] (SLE), in which the author identified four primary psychological challenges associated with chronic illness onset. These challenges included 1) increased depressive symptoms associated with loss of a past self, 2) increased depressive and anxiety symptoms associated with disease uncertainty, 3) increased physical and emotional fatigue, and 4) difficulties managing financial strain associated with long term illness. In concert with Beckerman's findings, other scholars (e.g. Bury 1991, Charmaz 1991, Siegel and Lekas 2002) have identified uncertainty about one's future as the primary way that chronic illness onset disrupts a person's self-concept and disarms them of mastery.

[10] SLE is a chronic autoimmune disease that is characterized by periods of fatigue, skin changes, and chronic inflammation of the joints and organs, primarily kidneys, which can lead to deterioration of organ function.

Other scholars (Bayliss et al. 2003, Hays et al. 1995, Yang 2006) have identified specific illness characteristics that contribute to how disruptive chronic illness is to an individual's coping and mental health. These illness characteristics, functional impairment and comorbidity, have received significant attention in the literature (Turner and Noh 1988) as influential moderating factors. Functional impairment, the degree of limitation in completing activities of daily living, has consistently been identified as a significant predictor of depressive symptoms in the literature (Turner and Noh 1988, Katon 2003). Among the chronically ill, decreased functioning predicts increased depressive symptoms and major depressive disorders that in turn, are associated with heightened perceived pain and limited functioning (Katon 2003, Kim et al. 2000). Regarding comorbidity, scholars (Bayliss et al. 2003) acknowledge that having multiple chronic physical health conditions significantly increases a person's risk for depressive symptoms and clinical depression diagnoses. As a result, the presumed increase in depressive symptoms after illness onset may depend on the number of comorbid physical health conditions. Findings that functional impairment and comorbidity influence the chronic illness – depressive symptoms relationship suggests that chronic illness is not a uniform experience across people or time.

In conclusion, a review of the literature has established that the chronic illness - depressive symptoms relationship exists among the young (Hobbie et al.2000), disparities in depressive symptoms exists between people with chronic illnesses and the healthy (Turner and Wood 1985), demographic differences in prevalence of depressive symptoms exist (Lincoln et al. 2010), and chronic illness functions as a stress proliferator by increasing likelihood of functional impairment (Park et al. 2004) and comorbidity (Kim et al. 2000).

Below, I link this review to a more thorough discussion of the competing explanations (cumulative disadvantage and youthful resilience) for how timing of chronic illness onset may impact mastery and depressive symptoms.

2.4 Cumulative Disadvantage versus Youthful Resilience

2.4.1 Cumulative Disadvantage

Cumulative disadvantage theory posits that early advantage or disadvantage situate a person for continued advantage or disadvantage at later stages of the life course (Ferrarro and Moore 2003). These gains or deficits are determined by one's individual characteristics, such as race, social class, health status, or ability, and the sociocultural assessment of those characteristics (Dannefer 2003). As some characteristics are less esteemed than others, individuals and groups are differentially positioned for disadvantage. This unequal assessment of characteristics and people leads to differences in social esteem, opportunity, and ultimately, position in the social hierarchy (Kutateladze et al. 2014). As a result, the social advantages or disadvantages experienced early in life structure the remainder of the life course by reproducing opportunity or challenges in multiple life domains (Shuey and Wilson 2008). Across the life course, this proliferation of advantage or disadvantage and the enduring effects of each collectively define a person's life trajectory and outcomes (Umberson et al. 2014).

Considered in tandem with the stress process (Aneshensel 1992, Pearlin 1981), the cumulative disadvantage explanation based on cumulative disadvantage theory (Ferrarro and Moore 2003) explains how chronic illness onset initiates the stress proliferation process and why trajectories of mastery and depressive symptoms may differ by health status and by timing of illness onset. This process of stress proliferation begins when an initial stressor (e.g. illness onset) produces additional stressors and strains in a person's life and within their social network

(Turner and Lloyd 1999). These additional stressors and strains can range in degree of disruption, duration of impact, and life area affected (Clark et al. 2002, Pearlin 2010). For example, in the case of chronic illness onset, financial strains due to work days missed or poor employment reviews (Vickers 2003) are evidence of the ways in which the chronically ill accumulate disadvantage when compared to healthy peers.

Moreover, the timing of the stressor is a critically important dimension to gauging its potential to cause harm and position people differently for loss of protective psychological resources (Elder et al. 1996, Hobbie et al. 2000, Pearlin 2010). The timing of chronic illness onset matters because the duration of its effects, namely the accumulation of disadvantage via the stress proliferation process, may differ depending on when in life illness onset occurs (Pearlin 2010).

As a protective coping resource, mastery increases with age at a decreasing rate and thus, the opportunity (i.e. time) to acquire more of this resource prior to illness onset is beneficial (Mirowsky and Ross 2003) in averting depressive symptoms. Consequently, illness onset at mid-life or late-life may position a person for lower depressive symptoms than someone who becomes ill as a young adult. In this study, I refer to this explanation as the "cumulative disadvantage explanation" referencing the idea that increased age at illness onset begets advantage. Below, I present an alternative to the cumulative disadvantage explanation, which I call youthful resilience.

2.4.2 Youthful Resilience

Resilience, "the ability of an individual to function competently in the face of adversity or stress" (Murphey et al. 2013) has been widely studied by developmental scholars that examine psychological outcomes of early trauma and disruptive life events (Karoly and Ruehlman 2006,

Norris et al. 2009). Much of this scholarship has highlighted how children and adolescents more effectively cope and ultimately, recover from traumatic or disruptive experiences than do adults (Easterbrooks et al. 2013, Karatsoreos and McEwen 2011) due to cognitive and psychological plasticity. This recovery is explained as being developmentally determined and related to children's more supple cognitive structure and less cemented self-concepts (Erikson and Erikson 1997, Wiebe et al. 2005). Developmental psychologists and neuropsychologists posit that plasticity, the greater flexibility and adaptability of the developing brain in early life in reaction to change (Stiles 2000), contributes to younger people's resilience when faced with trauma, physical disability, and chronic hardship (Davidson and McEwen 2012, Karatsoreos and McEwen 2011, Utwatte and Taub 2012). These findings suggest that resilience may be inversely related to age, meaning that younger people are more resilient in the face of disruptive life events than older people.

A youthful resilience explanation suggests that illness onset during an early developmental stage, as compared to mid-life or late-life, is less detrimental to a person's mastery and psychological coping because self-concepts are more malleable and challenges to self-concept, like functional impairment, are less threatening than during subsequent stages (Campbell-Sills et al. 2006). During the early life course, shifts in self-concept, independent of cause or outcome, are considered contributory not erosive to an established self (Arnett 2004). This age-related resilience may explain the rejection of this study's hypotheses (discussed below) and support that young adults fare better or as well in the preservation and mobilization of mastery and avoidance of depressive symptoms after illness onset than do healthy young adults and people who experience onset at later stages.

In conclusion, the theoretical frameworks of the life course perspective and stress process in conjunction with literature on chronic illness, mastery, and depressive symptoms provide context for answering this study's research questions, which were presented in the introductory chapter. Additionally, the competing explanations (cumulative disadvantage and youthful resilience) offer rationale for why subsets of people facing the same stressor may cope with its effects differently. In the section that follows, I will discuss areas in need of further study that I have identified as a result of reviewing the literature and outline hypotheses that were tested in this study.

2.5 Critique of Literature & Hypotheses

In review of the scholarly literature on chronic illness and its relationship to mastery and depressive symptoms, I have identified several areas requiring further study, which are discussed below and followed by the respective hypotheses that were tested.

Within the literature, some have argued that individual differences in psychosocial resources reflect individual variation in family background (Carlson and Corcoran 2001) or personality (Aldwin et al. 1996). However, I hypothesize that differences in these resources, specifically, mastery, reflect differences in exposure to stressful life events, like chronic illness onset, as informed by the stress process. In this study, I hypothesize that experiencing a disruptive life event early is especially harmful and impactful because the timing precludes people from adequately preparing themselves to manage the event's ramifications (Pearlin 2010). Moreover, the effects of chronic illness onset are particularly challenging to manage as they are chronically enduring, yet require continual adaptation with no expectation of full respite (Charmaz 1994). In this case, mastery takes on particular importance, as greater perceived control over circumstances is psychologically protective when faced with ever-changing

physical, mental, or social conditions (Pudrovska 2010). To review, this study's research questions are as listed:

1. What is the relationship between early onset chronic illness and mastery? [11]

2. What is the relationship between early onset chronic illness and depressive symptoms? Does mastery mediate the relationship between early onset chronic illness and depressive symptoms? [12]

3. Is early onset chronic illness (24-35) associated with lower mastery than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)?

4. Is early onset chronic illness (24-35) associated with greater depressive symptoms than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)? Does mastery mediate or moderate this relationship?

As a result of reviewing the literature, I expect the following:

- Hypothesis 1a. Early onset chronic illness is significantly associated with lower mastery compared to those without early onset chronic illness.

In this study, I conceptualize chronic illness onset to be a life event, a singular occurrence that initiates the transition in social status from healthy to the socially undesirable status of ill (Bury 1982). As a socially undesirable status, being a person with chronic illness results in deleterious short and long-term consequences (Hays et al.1995, Stroup et al. 2001, Simoni 2006). Scholars have repeatedly identified chronic illness as a significant risk for increased mental distress (e.g. Turner and Noh 1988); however, most studies examined this relationship exclusively among older adults (Pudrovska et al. 2005). Among these studies, mastery has been credited as a mediating mechanism by which health status impacts depressive symptoms (Miller

[11] Early onset refers to onset that occurs prior to mid-life and late-life, which in this study includes the ages of 36 -64 and 65 and older, respectively.

[12] Mediation is the process by which an independent variable (chronic illness) and dependent variable (depressive symptoms) establish an association through an intervening variable (mastery). Without this mediating variable, the significant bivariate relationship does not exist. In contrast, moderation is the process by which a variable (age at onset) changes the strength or direction of a relationship between a predictor (chronic illness) and dependent variable (mastery).

et al. 1995, Pearlin et al. 2007). In order to establish that these findings extend to young adults, the following hypotheses were tested:

- Hypothesis 2a. Early onset chronic illness is significantly associated with higher depressive symptoms compared to those without early onset chronic illness.
- Hypothesis 2b. Mastery mediates the relationship between early onset chronic illness and depressive symptoms.

At its core, this study is undergirded by the idea that stressors are contextually defined and their outcomes are contextually dependent. In this study of the effect of timing of chronic illness onset on mastery, the primary context in which this relationship occurs is age. No identified studies have examined if age at illness onset influences a person's availability of coping resources or explains age cohort differences in mental health outcomes. Through the present study, I apply the life course concept of timing in lives (Elder 1985) to the experience of chronic illness onset, which has not been addressed adequately in the literature. Additionally, earlier studies (Cott et al. 1999, Dickson et al. 2008) have not included young adults as a significant proportion of their chronically ill samples in order to test if the negative chronic illness- mastery relationship established among middle age and older adults is similar for the young. In order to examine if there are differences among chronically ill people in their development and retention of mastery depending on life stage at onset, I tested the hypothesis below:

- Hypothesis 3a. Early onset chronic illness is associated with lower mastery than illness onset at mid-life or late-life.

In this study, I hypothesize that chronic illness onset as a predictor of depressive symptoms differs depending on when in life illness occurs. The mental health outcomes

associated with chronic physical illnesses have been widely studied (Beckerman 2011, Kivuruusu et al., 2007, Ruehlman et al., 2010). Most literature, however, has consistently failed to acknowledge or examine potential differences between young adults, middle age adults, and the elderly. While scholars have often argued that increased depressive symptoms in late-life are due to declining physical health (Taylor and Lynch 2004), few have examined if the relationship between depressive symptoms and physical health is similar among non-elderly adults. Lastly, prior studies have not included young adults as a significant proportion of their samples (Hahn et al. 2014, Moen 2001) in order to test if the positive chronic illness- depressive symptoms relationship established among middle age and older adults is similar for the young. Due to literature (Chapman and Perry 2008, Fisk 2009) that suggests that older adults generally experience lower depressive symptoms than young adults, irrespective of health status, I expect the following:

- Hypothesis 4a. Early onset chronic illness is associated with greater depressive symptoms than illness onset at mid-life or late-life.

In reference to the cumulative disadvantage explanation presented earlier, I posit that age at time of chronic illness onset and disadvantage resulting from illness onset are inversely related, meaning that ill young adults are disadvantaged in mastery acquisition and/or retention when compared to healthy peers and people who experience onset later. Mastery's mediating role in the chronic illness- depressive symptoms relationship is established in the literature (Ormel et al. 1997, Nurullah 2010, Sacco et al. 2013), however, this relationship has typically been tested using exclusively middle age and elderly samples (Mausbach et al. 2012, Shnittiker et al. 2005). To examine if mastery's mediating function extends to young adults, I tested the hypothesis below:

- Hypotheses 4b. Mastery mediates the relationship between timing of illness onset and depressive symptoms.

Ill young adults' possession of less mastery than middle age and older adult chronically ill adults means that they are ill-equipped and thus, disadvantaged in their ability to manage negative outcomes. As suggested by cumulative disadvantage theory (Ferraro and Moore 2003), this relative disadvantage in possession of a critical coping resource may contribute to worse mental health, specifically greater depressive symptoms, than adults with onset during mid-life or late-life. In accordance with a life course perspective (Elder 1985), I conceptualize age as the primary context in which illness onset occurs. Thus, timing of illness onset may condition the relationship between chronic illness and depressive symptoms. No identified studies have investigated if mastery moderates the effects of timing of chronic illness onset on depressive symptoms. In order to investigate mastery's moderating effects on the timing of chronic illness – depressive symptoms relations, the following hypothesis was tested:

- Hypothesis 4c. Mastery moderates the relationship between timing of illness onset and depressive symptoms.

In conclusion, the hypotheses outlined above derive from areas that have not been adequately addressed in the current literature. Through analyses presented in later chapters, I aim to contribute to this body of knowledge regarding the effects of chronic illness onset on coping resources and mental health.

2.6 **Organization of Project**

In the chapter that follows (Chapter 3), I outline the methods used in testing the aforementioned hypotheses and present the strengths and limitations of the present study. In Chapter 4, I present analyses related to mastery as the outcome. Chapter 5 includes analyses and

findings regarding depressive symptoms. Lastly, in Chapter 6, I discuss the findings of the preceding chapters in the context of the cumulative disadvantage and youthful resilience explanations discussed earlier in this chapter and outline directions for future scholarship in the areas of chronic illness and life course studies.

3 METHODOLOGY

3.1 Introduction

In this methods chapter, I discuss specifics of study design, including a review of this study's research questions, the hypotheses to be tested, a description of the American Changing Lives study, sampling procedures, and descriptions of the samples used in analyses. Later, I outline the techniques I used in identifying and managing missing data, including multiple imputation. In the final sections of this chapter, I present detailed descriptions of each measure used in analyses and discuss the statistical method employed, multilevel modeling (MLM).

3.2 Study Design

3.2.1 Research Questions and Hypotheses

1. What is the relationship between early onset chronic illness and mastery?

    1a. Early onset chronic illness is significantly associated with lower mastery compared to those without early onset chronic illness.

2. What is the relationship between early onset chronic illness and depressive symptoms? Does mastery mediate[13] the relationship between early onset chronic illness and depressive symptoms?

    2a. Early onset chronic illness is significantly associated with higher depressive symptoms compared to those without early onset chronic illness.

    2b. Mastery mediates the relationship between early onset chronic illness and depressive symptoms.

3. Is early onset chronic illness (24-35) associated with lower mastery than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)?

[13] Mediation is the process by which an independent variable (chronic illness) and dependent variable (depressive symptoms) establish an association through an intervening variable (mastery). Without this mediating variable, the significant bivariate relationship does not exist. In contrast, moderation is the process by which a variable (age at onset) changes the strength or direction of a relationship between a predictor (chronic illness) and dependent variable (mastery).

3a. Early onset chronic illness is associated with lower mastery than illness onset at mid-life or late-life.

4. Is early onset chronic illness (24-35) associated with greater depressive symptoms than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)? Does mastery mediate or moderate this relationship?

    4a. Early onset chronic illness is associated with greater depressive symptoms than illness onset at mid-life or late-life.

    4b. Mastery mediates the relationship between timing of illness onset and depressive symptoms.

    4c. Mastery moderates the relationship between timing of illness onset and depressive symptoms.

3.2.2 Data

3.2.2.1 Data Description

The American Changing Lives Survey (ACLS) is a nationally representative longitudinal study of American adults 24 years of age and older. Due to the study's attention to the intersection of physical and mental health and psychosocial resources across the life course, the ACLS survey provides useful information for the examination of this study's research questions on the effect of timing of chronic illness onset on mastery and depressive symptoms. In this study, I analyze data from waves 1-4 of the American Changing Lives Survey. Survey administration for these waves occurred in 1986, 1989, 1994, and 2002.

3.2.2.2 Study Sample

American Changing Lives survey respondents were randomly identified and selected to participate from the American adult population 24 years of age and older. Those who agreed became the interview cohort at wave I of data collection in 1986. Ranging in age from 24 to 94 at wave 1, the ACLS sample is a nationally representative sample of American adults (n=3,617). Oversampling of black and older adult (60+) respondents yielded overrepresentations (2:1) of

these groups in the sample. During waves 1 and 2, surveys were exclusively administered through face to face interviews at respondents' homes. During waves 3 and 4, surveys were administered by telephone in addition to face to face interviews. Sample sizes and retention rates for each wave of data used in analyses (waves 1-4) are below.

Table 3.1 American Changing Lives Study Sample by Wave

Interview Year	1986	1989	1994	2002
N=	3617	2867	2562	1787
% Survivors Re-Interviewed From Prior Wave	---	83	80	80

In analyses, two samples were created, (1) the young adult sample and the (2) all ages restricted to chronically ill sample. Inclusion criteria for the young adult sample were as follows:

1. No reported chronic illnesses at wave 1
2. Age at wave 1 was between 24 and 35 years old
3. Item nonresponses were listwise deleted on dependent variables, mastery and depressive symptoms
4. Dropped four respondents that reported being multiracial
5. All respondents who dropped out after wave one were removed.
6. I removed those who attritted from one or more waves of data collection (though in early analyses I included them to assess how attrition affects the results).

The procedure for young adult sample selection is shown in figure 3.1 below.

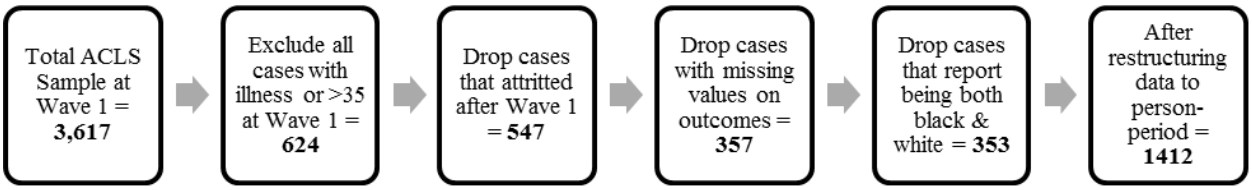

Figure 3.1 Procedure for Young Adult Sample Selection

The complete data sample of adults 24-96 consisted of 3,617 respondents. After excluding respondents older than 35 years of age at wave 1 and those with a chronic illness at wave 1, I reduced the sample size to 624 cases. Next, I dropped all cases that had only wave of

data and attritted out of the sample after baseline, reducing sample size to 547. I then dropped all cases with any missing values on dependent variables, mastery or depressive symptoms. Deleting these cases decreased sample size to 357. Four cases that racially self-identified as black and white were dropped from the sample since there were too few to be considered a separate racial category. I also decided that the assignment to either the black or white racial categories would be arbitrary. Dropping these cases (n=4) further reduced sample size to the final sample size of 353 cases, which when data was restructured became 1412 observations. Descriptive statistics for the young adult sample are included in table 3.2.

Table 3.2 Descriptive Statistics for Young Adult Sample (N=353)

Continuous Measures		**Wave 1**	**Wave 2**	**Wave 3**	**Wave 4**
		Mean (S.E.) St. D. [Range]			
Mastery	Early onset ill[14]	-1.34 (.154) 1.10 [-2.76 -1.09]	.061 (.155) 1.11 [-2.76- 1.09]	-2.75 (1.10) .930 [-2.75 -1.10]	-.191 (.134) .955 [-2.75 – 1.10]
	Healthy	.089 (.048) .827 [-2.76 -1.09]	.184 (.047) .812 [-2.76 -1.09]	.216 (.046) .792 [-2.75 -1.10]	.023 (.054) .940 [-2.75 – 1.10]
CESD	Early onset ill	.316 (.162) 1.15 [-1.11 – 3.94]	-.011 (.134) .958 [-1.11 – 2.44]	.039 (.165) 1.18 [-1.11 -3.42]	-.049 (.158) 1.13 [-1.11 -3.69]
	Healthy	-.011 (.056) .970 [-1.11 – 4.44]	-.197 (.047) .817 [-1.13 – 3.96]	-.359 (.046) .791 [-1.11 – 3.17]	-.309 (.050) .867 [-1.11 – 4.08]
Age	Early onset ill	27.9 (.343) 2.45 [24- 32]	30.9 (.343) 2.45 [27 – 35]	35.9 (.343) 2.45 [32 – 40]	43.9 (.343) 2.45 [40 – 48]
	Healthy	30.4 (.170) 2.96 [25 – 35]	33.4 (.170) 2.96 [28 - 38]	38.4 (.170) 2.96 [33 – 43]	46.4 (.170) 2.96 [41 – 51]
Education	Early onset ill	4.41 (.149) 1.06 [1 – 5]	---	---	---
	Healthy	3.86(.053) .926 [1 – 5]	---	----	---
Income	Early onset ill	24568 (2576) 18399 [3125 – 85230]	---	----	---
	Healthy	30833 (1056) 18354 [3125 – 85230]	---	---	---

Dichotomous Measures		N (%)[15]
Black	Early onset ill	14 (4%)
	Healthy	60 (17%)
Othrace	Early onset ill	1 (.28 %)
	Healthy	13 (3.68%)
White	Early onset ill	36 (10.2 %)
	Healthy	229 (64.9%)
Male	Early onset ill	20 (5.7%)

[14] Early Onset (N= 51), Healthy (N=302)

[15] Percentages were calculated as proportion of entire young adult sample (n/353)

	Healthy	138 (39.1%)
Married	Early onset ill	33 (9.4%)
	Healthy	103 (29.2%)

Table 3.3 Summary of ANOVAs among Young Adult Sample for Mean Mastery & Depressive Symptoms at Baseline

Mean Scores		**Sum of Squares**	**DF**	**Mean Square**	**F**	**Sig.**
Mastery	Between Groups	2.168	1	2.168	2.858	.092
	Within Groups	266.319	351	.759	---	---
	Total	268.487	352	---	---	---
CESD	Between Groups	4.670	1	4.670	4.696	.031
	Within Groups	349.087	351	.995	---	---
	Total	353.757	352	---	---	---

Table 3.3 includes results from analysis of variance (ANOVA) for outcome variables, mean mastery and mean depressive symptoms, at baseline among the young adult sample. Results illustrate that at a significance level of $p<.05$, mean mastery did not significantly differ between young adults who would later report early onset chronic illness and their same age peers who remained healthy throughout the study ($p=.092$). However, depressive symptoms were found to significantly differ between young adults with early onset chronic illness and healthy young adults ($p=.031$), meaning that even prior to illness onset, adults who eventually report chronic illness before age 36 differ in mean depressive symptoms from their peers who remain healthy.

The second analysis sample is restricted to chronically ill of all ages. Inclusion criteria for this restricted to chronically ill of all ages sample are as follows:

1. No reported chronic illnesses at wave 1
2. Chronic illness reported at wave 2,3, and/or 4

3. Item nonresponses were listwise deleted on dependent variables, mastery and depressive symptoms

4. Dropped two respondents that are multiracial

5. All respondents who dropped out after wave one were removed.

6. I removed those who attritted from one or more waves of data collection (though in early analyses I included them to assess how attrition affects the results).

The procedure for the chronically ill of all ages sample selection is shown in figure 3.2 below.

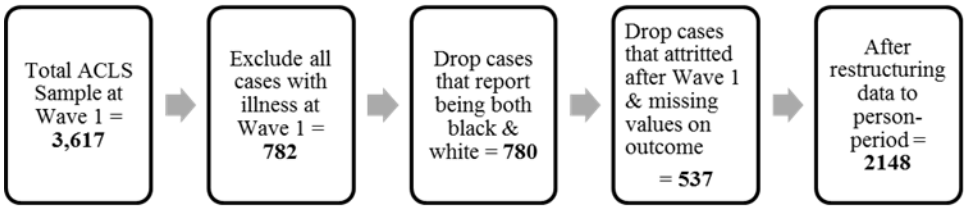

Figure 3.2 Procedure for the Chronically Ill of All Ages Sample Selection

The complete data sample of adults 24-96 consisted of 3,617 respondents. After excluding respondents with a chronic illness at wave 1, I reduced the sample size to 782 cases. Next, I dropped two cases that racially self-identified as black and white, reducing the sample to 780 cases. I dropped all cases that had only wave of data and attritted out of the sample after baseline and all cases with any missing values on dependent variables, mastery or depressive symptoms. Deleting these cases decreased sample size to the final sample size of 537 cases, which when data was restructured became 2148 observations. Descriptive statistics for the all ages restricted to chronically ill sample are included in table 3.4.

Table 3.4 Descriptive Statistics for All Ages Restricted to Chronically Ill Sample (N=537)

Continuous Measures		**Wave 1**	**Wave 2**	**Wave 3**	**Wave 4**
		Mean (S.E.) St. D. [Range]			
Mastery	Early[16]	-.134 (.154) 1.10 [-2.76 - 1.09]	.061 (.155) 1.11 [-2.76 – 1.09]	.139 (.130) .930 [-2.75 – 1.10]	-.191 (.134) .955 [-2.75 – 1.10]
	Mid	.053 (.047) .912 [-2.76 – 1.09]	.100 (.047) .920 [-2.76 – 1.09]	.162 (.045) .881 [-2.75 – 1.10]	-.090 (.052) 1.01 [-2.75 – 1.31]
	Late	.249 (.102) 1.04 [-2.76 - 1.09]	.328 (.094) .953 [-2.76 -1.09]	.306 (.091) .922 [-2.75 – 1.31]	-.033 (.098) 1.00 [-2.75 – 1.31]
CESD	Early	.316 (.161) 1.15 [-1.11 – 3.94]	-.011 (.134) .959 [-1.11 – 2.44]	.039 (.165) 1.18 [-1.11 – 3.42]	-.049 (.158) 1.13 [-1.11 – 3.69]
	Mid	-.021 (.052) 1.02 [-1.11 – 4.44]	-202 (.046) .901 [-1.11 – 3.78]	-.259 (.049) .954 [-1.11 – 4.74]	-.276(.044) .862 [-1.11 – 3.68]
	Late	-.495 (.062) .634 [-1.11 – 2.06]	-.402 (.067) .684 [-1.11 – 2.20]	-.486 (.062) .637 [-1.15 – 2.11]	-.312 (.077) .782 [-1.11 -2.11]
Age	Early	27.9 (.343) 2.45 [24 – 32]	30.9 (.343) 2.45 [27 – 35]	35.9 (.343) 2.45 [32 – 40]	43.9 (.343) 2.45 [40 – 48]
	Mid	40.1 (.438) 8.56 [25 – 61]	43.1 (.438) 8.56 [28 -64]	48.1 (.438) 8.56 [33 - 69]	56.12 (.438) 8.56 [41 – 77]
	Late	63.6 (.614) 6.26 [49 - 80]	66.6 (.4) 6.26 [52 – 83]	71.6 (.614) 6.26 [57 – 88]	79.6 (.614) 6.26 [65 – 96]
Education	Early	3.41 (.149) [1 – 5] 1.06	---	---	---
	Mid	3.65 (.053) 1.04 [1 – 5]	---	----	---
	Late	3.29 (.116) 1.19 [1 – 5]	---	----	---
Income	Early	24568 (2576) 18399 [3125 – 85230]	---	----	---
	Mid	32732 (1035.8) 20244 [3125 – 85230]	---	---	---
	Late	28507.8 (2104.7) 21464 [3125 - 82105]	---	---	---

[16] Early Onset (N= 51), Mid-life Onset (N= 382), Late-Life Onset (N=104).

Functional Impairment	Early	4.00 (.000) .000 [4 -4]	3.94 (.059) .420 [1 – 4]	3.92 (.062) .440 [1- 4]	3.82 (.092) .654 [1 -4]
	Mid	3.96 (.014) .268 [1 -4]	3.92 (.018) .350 [1 – 4]	3.82 (.031) .603 [1- 4]	3.71 (.038) .737 [1- 4]
	Late	3.90 (.040) .407 [1 -4]	3.88 (.031) .321 [3 -4]	3.84 (.058) .593 [1 – 4]	3.39 (.097) .989 [1- 4]
Comorbidity	Early	.00 (.000) .000 [0 -0]	.80 (.069) .491 [0 -2]	.53 (.090) .644 [0 -2]	.51 (.090) .644 [0- 2]
	Mid	.00 (.000) .000 [0 -0]	.36 (.031) .610 [0 – 3]	.75 (.043) .842 [0 – 7]	1.35 (.050) .971 [0 – 5]
	Late	.00 (.000) .000 [0 - 0]	.44 (.067) .680 [0 – 3]	.67 (.078) .794 [0 -4]	1.52 (.085) .870 [0 – 4]

Dichotomous Measures		N (%)[17]
Black	Early	14 (2.6%)
	Mid	83 (15.5%)
	Late	11 (2.1%)
Othrace	Early	1 (.19%)
	Mid	13 (2.4%)
	Late	0 (0%)
White	Early	36 (6.7%)
	Mid	386 (71.9%)
	Late	93 (17.3%)
Male	Early	20 (3.7%)
	Mid	148 (27.6%)
	Late	41 (7.6%)
Married	Early	33 (6.2%)
	Mid	248 (46.2%)
	Late	63 (11.7%)

[17] Percentages were calculated as proportion of entire all ages restricted to chronically ill sample (n/537)

Table 3.5 Summary of ANOVAs among All Ages Restricted to Chronically Ill Sample for Mean Mastery & Depressive Symptoms at Baseline

Mean Scores		Sum of Squares	DF	Mean Square	F	Sig.
Mastery	Between Groups	5.537	2	2.768	3.019	.050
	Within Groups	489.639	534	.917	---	---
	Total	495.176	536	---	---	---
CESD	Between Groups	27.226	2	13.613	14.505	.000
	Within Groups	501.165	534	.939	---	---
	Total	528.391	536	---	---	---

Table 3.5 includes results from analysis of variance (ANOVA) for outcome variables, mean mastery and mean depressive symptoms, at baseline among the all ages restricted to chronically ill sample. Results illustrate that at a significance level of $p<.05$, mean mastery did not significantly differ between subsets of adults who would later report chronic illness early, at midlife, and at late life. However, this statistical significance was marginal ($p=.05$). Alternatively, depressive symptoms were found to significantly differ between timing of chronic illness onset groups ($p=.000$), meaning that even prior to illness onset, adults who eventually report chronic illness differ in mean depressive symptoms at baseline by timing of illness onset.

3.2.3 Measurement

Analyses that used data from the young adult sample incorporated the following variables; time, chronic illness, depressive symptoms, mastery, age, race, sex, educational attainment, and income. In the section that follows, I discuss my conceptualization and operationalization of each of these variables.

3.2.3.1 Time

Time was measured as number of years since baseline interview. For all respondents, baseline interview occurred in 1986. Consequently, time equaled 0 for interviews in 1986. Time

equaled 3 for interviews in 1989. Time equaled 8 for interviews in 1994 and time equaled 16 for interviews in 2002.

3.2.3.2 Chronic Illness

Chronic illness, the enduring subjective experience of illness that is initiated by onset of symptoms of disease that are not expected to remit during an individual's lifetime (Bury 1982), was constructed and measured as a categorical variable in which respondents were designated as *chronically ill* (1) or *healthy* (0) based on their response to "Has respondent had a chronic health condition(s) in the past 12 months?" A response of yes at wave 2, 3, or 4 resulted in classification as chronically ill. In order to be classified as healthy, respondents must have reported no at waves 2, 3, and 4.

3.2.3.3 Depressive Symptoms

Depressive symptoms was measured using the Center for Epidemiologic Studies Depression (CESD-SF) Scale, which is included in all waves of ACLS data and has been used widely in social science research (Radloff 1977, Yang 2006). This measure was created and standardized in the original ACLS study data. The CESD (11-item) instrument measures the number and severity of depressive symptoms included as diagnostic criteria in the American Psychiatric Association's Diagnostic and Statistical Manual-V (2013). The CESD is administered as part of the ACLS survey and includes the following items:

(Please tell me how often you felt this way during the past week...)

1. R felt depressed.
2. R felt that everything R did was an effort.
3. R's sleep was restless.
4. R was happy.
5. R felt lonely.
6. R felt people were unfriendly.
7. R enjoyed life.
8. R did not feel like eating. R's appetite was poor.

9. R felt sad.
10. R felt that people did not like R in the past week.
11. R could not get "going."

Each of these items is an ordinal variable with three Likert response categories ranging from 1 (“rarely, none of the time”) to 3 (“most, all of the time, 5-7 days”). Items 4 and 7 were reverse coded. Consequently, this study’s measure of depression equaled a respondent’s average value across the 11 items. Response values for the CESD scale ranged from 11 to 33, with higher values signifying greater depressive symptomatology. This measure was then standardized so that the mean score was centered at 0. Consequently, this study’s measure of depression reflects deviation from the sample mean score of depressive symptoms, with positive values representing depressive symptom scores above the mean and negative values representing scores below the mean.

The CESD is routinely used in clinical research (Frech et al. 2011), community based psychiatric research (Goodman and Must 2011), and social science research (Cohen et al. 1993, Rooks et al. 2011) to establish a likely diagnosis of depression, which is consistently validated by a professional’s assessment (Millette et al. 2010, Olino et al. 2012). Moreover, like the self-administered CESD, ACLS survey items about physical illnesses rely exclusively on respondents’ self-reports and do not inquire about the source of diagnosis.

3.2.3.4 Mastery

Mastery, one’s perceived ability to control and cope with life circumstances (Pearlin et al. 2007), was measured as a continuous variable using The Pearlin Mastery Scale, which is included in all waves of American Changing Lives data. This measure was created and standardized in the original ACLS study data. Previous studies (e.g. Maslow et al. 2011) have similarly measured mastery. The seven items included on The Pearlin Mastery Scale include:

1. No way I can solve some of the problems I have.
2. Sometimes I feel that I am being pushed around in life.
3. I have little control over the things that happen to me.
4. I can do just about anything I really set my mind to.
5. I often feel helpless in dealing with the problems of life.
6. What happens to me in the future mostly depends on me.
7. There is little I can do to change many of the important things in my life.

Each of these items is an ordinal variable with four Likert response categories (4= strongly disagree to 1=strongly agree). Items 4 and 6 were reverse coded, so that on these items higher scores represent lower mastery. Consequently, this study's measure of mastery equaled a respondent's average value across the 7 items. Response values for the mastery scale ranged from 7 to 28, with higher values signifying a greater sense of mastery. This measure was then standardized so that the mean score was centered at 0. As a result, this study's measure of mastery represents deviation from the sample mean score of mastery, with positive values representing scores above the mean and negative values representing scores below the mean.

3.2.3.5 Demographic Characteristics

Respondent's current age was measured using the survey item, "Age of respondent (in years) at date of interview." As this variable was only included on the baseline survey, I constructed age variables for the subsequent waves by adding the number of years since baseline to age or respondent at baseline. For example, a respondent that was 25 in 1986 was assigned a value of 28 in 1989, 33 in 1994, and 41 in 2002. Respondent race was measured with two nominal variables, "race of respondent - black" and ""race of respondent – white." Response categories were treated as mutually exclusive, meaning cases that identified both white and black race were deleted from the sample. Responses of being non-white and non-black were aggregated into the category of "other race."

Sex was measured using the nominal variable, biological sex at wave 1 (1986). This dichotomous variable included categories of male and female (reference). Education was operationalized as an ordinal variable, using the variable, "highest grade of school completed as of interview (1986)," which included response categories ranging from 0 (none) to 20 (8 years of college or more). I recoded and collapsed these categories into five categories of an 8th grade education or less, some high school, GED/high school graduate, some college/post-secondary, and college graduate. Income was measured as a continuous variable using the ACLS created and standardized measure, "1986 family income." This variable was adjusted to include ten categories that represented the midpoints of each $10,000 income range represented in the sample.

Analyses that used data from the all ages restricted to chronically ill sample incorporated all of the variables above and three additional variables; timing of chronic illness onset, functional impairment, and comorbidity. Below, I discuss my conceptualization and operationalization of these additional variables.

3.2.3.6 Timing of Chronic Illness Onset

Timing of chronic illness onset was measured as two dichotomous variables indicating group membership. Cases that were healthy at baseline and subsequently reported a chronic health condition at any subsequent wave (waves 2-4) were included in the all ages restricted to chronically ill sample. From this sample, subgroups by age at onset were created in the following manner:

1. Illness at time 1 and Age 24-35 at time 1 or Illness at time 2 and Age 24-35 at time 2 or Illness at time 3 and Age 24-35 at time 3 = <u>Early Onset</u>
2. Illness at time 1 and Age 36 – 64 at time 1or Illness at time 2 and Age 36 – 64 at time 2 or Illness at time 3 and Age 36 – 64 at time 3 = <u>Mid-Life Onset</u>

3. Illness at time 1 and Age 65 and older at time 1 or Illness at time 2 and Age 65 and older or Illness at time 3 and 65 and older at time 3 = Late-Life Onset

In analyses, mid-life and late-life onset served as categories of interest and early onset served as the reference category.

3.2.3.7 Illness Characteristics

Functional impairment in the areas of physical work and daily living were measured using a functional impairment index created and standardized in the original ACLS data. This index is composed of the following items:

1. In bed/ chair most or all day due to health/ has a lot of difficulty or cannot bathe self.
2. Has a lot of difficulty or cannot climb a few flights of stairs or walk several blocks because of health.
3. Has a lot of difficulty or cannot do heavy work around the house such as shoveling snow or washing walls because of health.
4. Does not have a lot of difficulty doing heavy work around the house such as shoveling snow or washing walls because of health.

Each of these survey items was originally measured ordinally (1= no impairment to 4= significant impairment). Item 4 was reverse coded. Survey items were then aggregated into the functional impairment index. For this pre-created index, responses were categorized into categories of no impairment (4), moderate impairment (3), minimal impairment (2), and high impairment (1). Thus, larger values equal less functional impairment. Diagnostic comorbidity was measured using the following survey item, "Number of chronic health conditions." This continuous variable included responses ranging from 0 to 9 conditions.

In the section that follows, I discuss my decision making process in managing missing data due to across wave attrition. Analyses of missing data and subsequent, analytic decisions pertaining to that missing data are discussed in detail in the section below.

3.2.4 Missing Data

3.2.4.1 Assessing Missing Data

In order to identify patterns and predictors of missing data, I created a dummy variable, "missing," in which all cases that attritted after baseline were assigned a value of 1 and all variables with complete data on outcome variables were assigned a value of 0. I then ran logistic regressions to assess if missingness was associated with the study outcomes, mastery and depressive symptoms. I first investigated missingness within the young adult sample by examining the bivariate relationships between mastery and missingness (table 3.4, model 1) and depressive symptoms and missingness (table 3.4, model 2). Note that there are 353 valid non missing cases and 194 cases with missing information on one or more waves for a total of 547 cases in this set of analyses.

Table 3.6 Logistic Regression - Predictors of Missingness (Young Adult Sample)

(N=547)	Model 1	Model 2	Model 3
Intercept	-1.05 (.098) [---]	-1.06 (.098) [---]	.497 (.328) [---]
Mastery	-.276* (.138) [.759]	---	-.251 (.138) [.778]
CESD	---	.159 (.099) [1.17]	-.062 (.124) [.940]
Male[a]			.223* (.107) [1.56]
Black[b]			.620*** (.0157) [3.46]
Othrace[c]			.468* (.187) [2.53]
Income			-6.97^{-6} (6.97^{-6}) [1.00]
Married[d]			-.317** (.119) [.530]
Educ<HS[e]			1.309 (.711) [7.19]
Educ- Some HS[f]			.077 (.319) [2.10]
Educ- HS Grad[g]			-.326 (.243) [1.40]
Educ – Some College[h]			-.40 (.249) [1.31]
-2LL	571.735	619.450	571.735
ΔD[18]	---	47.72**	47.72**

***p<.001 ** p< .01 * p<.05

Note: raw logit confidents are presented first along with standard errors in parentheses and odds ratios in brackets. [a] reference category is: female. [b, c] reference categories are: white. [d] reference category is: unmarried. [e-h] reference categories are: college graduate.

In model 1, among young adults, mastery was significantly and negatively associated with missing data (b=-.276, p<.05). Young adults with higher mastery scores were approximately 24% less likely to drop out of the sample than those with lower mastery. The

[18] ΔD denotes the change in deviance from one model to the subsequent model. The values presented represent the change in -2LL and its significance when compared to the X^2 value that corresponds with the difference in degrees of freedom from a model to the next model.

young adult sample is thus overrepresented by cases with higher mastery and findings derived from this sample are not representative of the entire American young adult sample (24-35) in 1986. Estimates later presented in chapters 4 and 5 must be interpreted as conservative and reflective of the limitations of this sample, specifically, the bias towards adults with higher mean mastery and underrepresentation of young adults with lower levels of personal mastery.

In model 2, among young adults, depressive symptoms were not a significant predictor of missingness. The sample is not biased by an unrepresentative proportion of cases with high or low CESD scores. Estimates later presented in chapters 4 and 5 should be interpreted as nationally representative, with the caveat that mastery as a mediating or moderating factor is biased, as previously discussed. Below, I present results from the final model predicting missingness among young adults (table 3.4, model 3). In this model, I included socioeconomic and demographic characteristics to assess what factors contribute to a case's likelihood of missing data.

Once controlling for the effects of socioeconomic and demographic characteristics and depressive symptoms, mastery was no longer a significant predictor of missingness. However, sex, race, and marital status were significantly associated with missing data. Men were 1.6 times more likely than women to be missing from the sample ($p<.05$). Black respondents were 3.5 times more likely than white respondents to drop out of the study sample ($p<.001$). Young adults reporting another racial category were 2.5 times more likely than white peers to be missing from the sample ($p<.05$). In the final analytic models presented in chapters 4 and 5, all socioeconomic and demographic characteristics were included to adequately control for their effects on the respective outcomes, yet caution will still be taken in interpreting all findings.

Next, I extended the assessment of missing data to the all ages restricted to chronically ill sample and ran logistic regression models to examine if either of the outcome variables, mastery or depressive symptoms, was associated with missing data. Results of these analyses are presented below in table 3.5. Note that there are 537 valid non missing cases and 243 cases with missing information on one or more waves for a total of 780 cases in this set of analyses.

Table 3.7 Logistic Regression - Predictors of Missingness (All Ages Restricted to Chronically Ill Sample)

(N=780)	**Model 1**	**Model 2**	**Model 3**
Intercept	-1.26*** (.086) [---]	-1.25*** (.086) [---]	-.621 (.356) [---]
Mastery	-.141 (.090) [.869]	---	-.115 (.106) [.892]
CESD		.062 (.088) [1.06]	-.108 (.106) [.898]
Male[a]			.131* (.099) [1.30]
Black[b]			.358* (.145) [2.05]
Othrace[c]			-.168 (.299) [.715]
Income			-5.49^{-6} (5.55^{-6}) [1.00]
Married[d]			-.106 (.109) [.809]
Educ<HS[e]			1.62*** (.294) [11.54]
Educ- Some HS[f]			.109 (.218) [2.56]
Educ- HS Grad[g]			-.278 (.158) [1.74]
Educ – Some College[h]			-.614*** (.187) [1.24]
-2LL	822.573	824.516	752.357
ΔD	---	1.94	72.16**

***p<.001 ** p< .01 * p<.05

Note: raw logit confidents are presented first along with standard errors in parentheses and odds ratios in brackets.

[a] reference category is: female. [b, c] reference categories are: white. [d] reference category is: unmarried. [e-h] reference categories are: college graduate.

Within the all ages restricted to chronically ill sample, mastery is not significantly associated with missingness. This finding means that the sample is not over or underrepresented by any particular range of scores on the Pearlin Mastery Scale. Similarly, as illustrated in table 3.5 (model 2), depressive symptoms are also not significant predictors of missingness among this sample.

In analyses presented in table 3.5 (model 3), I included socioeconomic and demographic characteristics and both outcomes as predictors of missingness. In this final model, neither outcome was significantly associated with missing data. Race and educational attainment were significantly associated with missingness. Black respondents were 2 times more likely than white respondents to be missing from the sample after baseline ($p<.05$). Respondents with the lowest level of education (less than 9th grade) were 11.5 times more likely to be missing than college graduates and respondents with some college education were 1.2 times more likely to be missing than college graduates ($p<.001$). Due to these demographic predictors of missingness, this sample is overrepresented by white adults and women. In all final analytic models presented in chapters 4 and 5, I include these characteristics in order to control for their effects.

3.2.4.2 Multiple Imputation of Missing Data

In order to ensure that there were no significantly differences in results from analyses using the listwise deleted datasets, I also multiply imputed the data sets and ran the same analyses on them to compare findings. Before discussing those findings, I will briefly discuss multiple imputation. Multiple imputation refers to any method in which computerized predicted values are inserted into the data as replacements for the missing data via regression methods that randomly draw data values and parameters (Allison 2002, 85). Multiple imputation is most

appropriate when 1) number of cases lost to listwise deletion is intolerable, 2) data is missing completely at random or missing at random, 3)a linear or nonlinear model is being estimated, 4) sample size is large, and 5) variables in the analytic model have a normal multivariate distribution (Allison 2002).

In cases in which data are not missing completely at random (MCAR) or missing at random (MAR), as in the case in this particular study, scholars (Allison 2000, Allison 2002, Rubin 1996) warn against using multiple imputation because it requires the researcher to make assumptions about the cause and pattern of missingness without the ability to test these assumptions. Specifically, Allison (2002; 86) argues that imputation requires sound and thorough "apriori knowledge of the mechanisms for generating the missing data," without which the arrival at stable and accurate estimates are improbable.

Logistic regression analyses presented above confirm that these data are not missing at random, but also not completely predicted by outcomes. Specifically, within the young adult sample, missingness is predicted by mastery score, however, demographic characteristics of race, sex, and marital status drive missingness not mastery itself. Within the all ages restricted to chronically ill sample neither outcome is significantly related to missingness, however, missingness is predicted by race, sex, and educational attainment.

Alternatively, listwise deletion is appropriate for use with most data when the patterns and predictors of missingness are identifiable. Allison cautions that limitations of listwise deletion include 1) reduced sample size and thus, power and 2) potential bias in representativeness of sample, while a strength of this method is less opportunity for researcher error and introduction of bias into the model (2002). Specifically, Allison regards the lack of

manipulation of data as the greatest strength of listwise deletion, as artificial and potentially inaccurate estimates are risks of all other conventional methods for handling missing data.

For the sake of demonstration and comparison, I multiply imputed data sets for the young adult sample and all ages restricted to chronically ill sample using the Proc MI and Proc MI Analyze procedures embedded in SAS 9.3. All predictors that appear in the analytic models were included as predictors of missingness. Those tables can be found in appendix A.[19] For the young adult sample, there were no major analytic differences in predicting either mastery or CESD using the listwise sample versus using the multiply imputed sample. For the chronically ill of all ages sample, there is one notable difference between the listwise deletion sample and the multiply imputed sample This process, its results, and my final analytic decisions are discussed in the section that follows. Discussion of these results in relation to final results presented in chapters 4 and 5 is also included below.

Just as in the case of models of mastery among the young adult sample using listwise deletion, mastery is most appropriately modeled by the inclusion of a linear and quadratic term (table 7.1). Fixed effects estimates remain very similar to those using listwise deletion in effect size, direction, and significance. The only noted difference was in the significance of educational attainment less than high school, which is significant with listwise deletion and non-significant after imputation (table 7.2) .

As in the case of models of mastery among the all ages restricted to chronically ill sample using listwise deletion, mastery is most appropriately modeled by the inclusion of a linear and

[19] In Appendix A, I have included tables that mirror analyses presented later in chapters 4 and 5. The appended tables present model fit statistics (table 7.1) and results (table 7.2) from 2-level MLM on mastery among the young adult sample and model fit statistics (table 7.3) and results on mastery among the all ages restricted to chronically ill sample (7.4). Subsequently, I present model fit statistics (table 7.5) and results (table 7.6) from 2-level MLM on depressive symptoms among the young adult sample and model fit statistics (table 7.7) and results (table 7.8) among the all ages restricted to chronically ill sample.

quadratic term (table 7.3). Fixed effects estimates overwhelmingly remain similar in effect size, direction, and significance across models 1-4 (table 7.4). However, there are important differences on key variables, late-life onset, other race, and function that are explained by the previously discussed bias in the sample, the underrepresentation of cases with the lowest levels of mastery.

In results from listwise deletion (table 4.4), late-life onset was not a significant predictor of mastery, meaning no statistical difference between late-life and early onset was detected. Yet, after imputation, the relationship is highly significant ($p<.001$). Similarly, reporting an "other race" and functional impairment were not significant in analyses with listwise deletion, but become significant after imputation. Estimates remain very similar for all three variables. The statistically significant differences in mastery between adults with late-life onset and those with early onset and adults with varying degrees of impairment are likely unidentifiable because there is less variation in mastery when cases are deleted listwise.

In table 7.5, results confirm that depressive symptoms among young adults are best modeled as curvilinear with the inclusion of the linear and quadratic terms. Fixed effects estimates (table 7.6) closely reflect those presented in results from listwise deletion (table 5.2). The main difference is the significance of black race in model 5 increases from the $p<.05$ to $p<.001$ level after imputation. Additionally, income is non-significant in the listwise deletion results, but highly significant ($p<.001$) in the imputed model.

Fixed effects presented in table 7.8 overwhelmingly reflect estimates resulting from analyses with listwise deletion (table 5.4). However, there are notable discrepancies in regards to a few variables. The most significant discrepancy occurs with mid-life onset which remains similar in effect size and significance between the two samples, but reverses sign. Specifically,

with listwise deletion, mid-life onset is associated with lower depressive symptoms than early onset, but after imputation, mid-life onset is associated with higher depressive symptoms than younger peers. This discrepancy is interesting but not surprising since, as previously noted; the listwise deletion sample includes an overrepresentation of cases with higher levels of mastery for every subsample (early, mid, late life onset). Using this method, distinctions between early and mid-life are not apparent because each subsample's "worst off" are absent, making them more similar. Also, after imputation, comorbidity becomes a significant predictor of depression ($p<.01$), likely reflecting that cases with lowest mastery now reinserted into the sample may also be those with more conditions.

Although there were a few, but noteworthy, discrepancies between results from the samples derived from listwise deletion versus samples derived from multiple imputation, overwhelmingly estimates were consistent across both methods of managing missing data. Ultimately, I proceeded with the non-imputed data that resulted from listwise deletion. Proceeding with caution of interpretation and recognizing the limitations of representativeness, listwise deletion offered the best method for managing these missing data because 1) sample size was relatively small, 2) data are not missing completely at random nor missing at random, and 3) the mechanisms that undergird the missing variables, mastery and depressive symptoms, specifically among chronically ill people, are not fully known. Lastly, additional advantages of listwise deletion include 1) its applicability to any statistical analytic technique, including multilevel models, and 2) ease of use with statistical programs (Allison, 2002). In the section that follows, I describe the method used in analyses of these data and testing of the aforementioned hypotheses.

3.2.5 Method

3.2.5.1 Description

Multilevel linear models (MLM) are useful for analyzing hierarchal data, data that includes variables that are nested within other variables. For example, in this study, repeated measures of mastery are nested in the individual respondent or case. Thus, the respondent is the contextual variable within which the individual mastery or depressive score at each time point is nested. In a repeated measures design, residuals are correlated because measures at each time point are influenced by the fact that they are derived from the same individual or context. As a result, the assumption of other linear models that errors are independent is violated.

Multilevel linear modeling (MLM) is the most appropriate method for analyzing longitudinal data in which data is nested within higher order data. MLM is an appropriate analytic technique for examining the study's research questions because 1) study data are hierarchal and longitudinal, 2) repeated measures designs violate general liner models' assumption of independent errors, and 3) these models are robust even when data is missing at one or more time points for a respondent.

3.2.5.2 Assumptions of Multilevel Linear Models

The assumptions of multilevel models are the same as those for general linear models with some modifications. Assumptions of the general linear model include 1) additivity and linearity, 2) normality of data, 3) homogeneity of variance, and 4) independence of error terms. As previously stated, a repeated measures design violates the general linear model assumption of independence of error due to auto-correlated data. Thus, a multilevel model is more appropriate than other methods, namely ordinary least squares regression, because MLM can correct for this lack of independence.

In order to account for non-independent observations, I measured the dependency in the data by calculating the intra-class correlation coefficient. An intra-class correlation (ICC) coefficient estimates how much of the total variance occurs between-persons versus within-person. For example, in this study using a two-level hierarchal data structure, where the level 1 variable is a repeated measure of mastery or depressive symptoms, I used the intra-class correlation coefficient to assess how much of the total variability in each outcome was attributable to the individual. The ICC also estimates how mastery and CESD scores at baseline are related to mastery and CESD scores at a subsequent time point.

3.2.5.3 Fixed and Random Effects

In contrast to general linear models, multilevel models allow for random parameters, values that can vary. One of the most useful features of multilevel models is that intercepts and slopes are not assumed to be fixed or equal for the entire sample. As a result, the baseline of a measure (intercept) and rate of growth or change (slope) in that measure over time can differ for every case in a sample. Although it is possible to model data with a random intercept and fixed slope or a fixed intercept and random slope, I modeled the data allowing for both random intercepts and random slopes. In these types of models, individuals' levels of mastery and depressive symptoms at baseline were allowed to vary, as were their rate of decrease or increase across subsequent time points. I decided on a random intercepts and random slopes model as this study examines individual psychological coping and mental health that are likely to differ across individuals and across time. As this study incorporated modeling of both random effects and repeated measures, an unstructured covariance structure was fitted. An unstructured covariance structure is commonly used for repeated measures design, particularly, growth models due to its

flexibility. In an unstructured covariance structure, the relationships between variances or scores are allowed to vary (Singer and Willett 2003).

3.2.5.4 The Multilevel Model

The *random slope and random intercept model* can be represented by three basic regression equations. The level 1 regression equation gives the deviation from a population mean (fixed effects) and appears below:

$$\mathbf{Y_{ij} = \beta_{0j} + \beta_{1j}X_{1j} + \beta_{2j}X_{2j} + e_{ij}}$$

Where Y_{ij} represents the outcome variable, mastery or depressive symptoms, for each person i at time j. $\beta 0j$ represents the intercept. β_{1j} represents the regression coefficient for the slope for the variable X_1. β_{2j} represents the slope for the second predictor variable, X_2. and e_{ij} represents the error term or residual.

The level 2 regression equations collectively estimate an individual's deviation on an outcome across time (random effects). These equations appear below:

$$\boldsymbol{\beta_{0j} = \gamma_{00} + \gamma_{01}W_j + u_{0j}}$$

$$\boldsymbol{\beta_{1j} = \gamma_{10} + u_{1j}}$$

Where β_{0j} denotes the intercept for the outcome at time j, γ_{00} represents the grand mean of the intercepts for a particular respondent when all other predictors are held constant. γ_{01} represents the slope of the outcome for the level 2 independent variable, W_j. u_{0j} refers to the error or residual at level 2. β_{1j} denotes the slope or rate of change in the outcome for the individual.

3.2.5.5 Model Fit & Interpretation

The overall model fit of multilevel models is evaluated by using a chi-square likelihood ratio test. Although multiple versions of the log-likelihood value exist, I primarily used Akaike's Information Criterion (AIC) and Schwarz's Bayesian Criterion (BIC). AIC is a goodness of fit

measure that corrects for the complexity of a model. The AIC estimates how much information is retained versus lost with a particular set of predictors in the model (Burnham and Anderson 2004). Comparable to the AIC, BIC is a more conservative goodness of fit measure that more strictly adjusts for the number of estimated parameters in a model. Thus, BIC is useful in avoiding the over fitting of models (Burnham and Anderson 2004). For each of these criteria, a small value denotes better fit (Field 2012). In this study, I built nested models and relied on the change in the AIC, BIC and -2LL values to compare models. -2LL is a measure of error or unexplained variation and a large value indicates poor model fit. When deciding on model fit, I calculated the change in deviance or -2LL (ΔD) from one nested model (e.g. model 1) to the subsequent model (e.g. model 2). This value was then compared to the critical value denoted on the Chi Square distribution that corresponded to the difference in degrees of freedom from a model (e.g. model 1) to the next model (model 2). Values greater than the X^2 critical value were evidence of significance at the $p<.05$ and $p<.01$ levels, which indicate improvement of model fit.

Estimates from multilevel models should be interpreted as those that derive from other regression models, such as ordinary least squares. In this study, I present estimates for fixed effects and random effects for each set of analyses. Fixed effects should be interpreted as population estimates, while random effects should be interpreted as average deviation for an individual from the mean. In chapters 4 and 5 that follow, I present analyses and results that aim to address the primary research questions that were outlined at the beginning of this chapter.

4 RESULTS: MASTERY

4.1 Analyses: Mastery as Outcome

In analyses detailed below, I fitted two-level random intercepts and slopes models using maximum likelihood estimation, in which I allowed both intercepts and slopes to vary across individuals. In the case of change in a personal coping resource, mastery, it is most reasonable to assume that individuals differ in their level of mastery at baseline and rate of change across a 16 year period. Thus, a random intercepts and slopes model is most appropriate for examining the primary research questions of this study.

In all analyses, I model the effects of 1) early onset chronic illness and 2) timing of chronic illness onset across 4 time points (16 years) on mastery. Time was measured to reflect the passage of time since first interview at wave 1 in 1986. Subsequently, time was measured as 0 (1986), 3 (1989), 8 (1994), and 16 (2002). Time also serves as a measurement proxy of aging. In all analyses, two random effects were modeled, in which I allowed intercept and slope to vary at each time point. Excluding respondent race, income, marital status, educational attainment and sex, all predictors were entered into the models as time varying.

After assessing multiple covariance structures, I decided that an unstructured covariance structure was most appropriate, as baseline estimates of mastery and its growth are strongly correlated. Additionally, an unstructured covariance structure allowed for random effects for mastery and at 0 (intercepts) and time (slopes), and the interaction between baseline mastery and rate of change (Littell et al. 2000).

Below, I present research questions and related hypotheses that pertain to this chapter's focus on mastery as the outcome.

4.2 Research Questions and Hypotheses:

Research Question #1: What is the relationship between early onset chronic illness and mastery?

Hypothesis 1a: Early onset chronic illness is significantly associated with lower mastery compared to those without early onset chronic illness.

Research Question #3: Is early onset chronic illness (24-35) associated with lower mastery than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)?

Hypothesis 3a: Early onset chronic illness is associated with lower mastery symptoms than illness onset at mid-life or late-life.

4.3 Results: Effects of Early Chronic Illness on Mastery

Table 4.1 presents four nested models estimating continuous mastery among the young adult sample (N= 1412). First, I fitted an "empty model" to estimate how much of the variation in mastery among the young adult sample occurs between-subjects. The intra-class correlation (ICC) of .3246 suggests that approximately 32.46 % of variation in mastery is attributable to between-person factors. Next, I estimated mastery trajectories by including a linear term (time) as a fixed effect only in model 2 and as fixed and random effects in model 3. Lastly, I included a quadratic term as fixed and random effects into model 4, allowing for random intercept and slope. In assessing model fit, -2 log likelihood, AIC, and BIC values were compared in deciding if model fit was significantly improved by inclusion of the quadratic term (time squared). As evidenced in table 4.1, the quadratic form best fit the data. Estimates for -2LL steadily decreased across models 1 through four (3626.5, 3615.2,3559.6, 3507.0).

Change in mastery among the young adult sample is curvilinear and convex (figure 4.1). Figure 4.1 illustrates that mastery among young adults at baseline hovers very slightly above average (.1), approached .2 at its peak around the study midpoint (year 8), and subsequently decreased and approached the mean by year 16. Thus, growth in mastery over the 16 years study

period was appropriately controlled for in every model before the introduction of independent or control variables. Tests of nested models (ΔD) revealed that each subsequent model (table 4.1) was an improvement on the model nested within it. Based off of the AIC (3527.0), BIC (3565.7), and tests of nested model (ΔD) values, model 4, which includes time and time squared as both fixed and random effects, was the superior fitting model.

4.3.1 Fixed Effects

Table 4.1 Model Fit Statistics on Mastery among Young Adults (N=1412)

(N=1412)	Model 1	Model 2	Model 3	Model 4
	Mastery	Mastery, Time (fixed)	Mastery, Time (fixed & random)	Mastery, Time (fixed & random), Time_sq (fixed and random)
AIC	3632.5	3623.2	3571.6	3527.0
AICC	3632.5	3623.3	3571.6	3527.2
BIC	3644.1	3638.7	3594.8	3565.7
-2LL	3626.5	3615.2	3559.6	3507.0
Chi Square	283.08***	287.29***	342.98***	381.92***
ΔD[20]	---	11.3**	55.6**	52.6**

***p<.001 ** p< .01 * p<.05

[20] ΔD denotes the change in deviance from one model to the subsequent model. The values presented represent the change in -2LL and its significance when compared to the X^2 value that corresponds with the difference in degrees of freedom from a model to the next model.

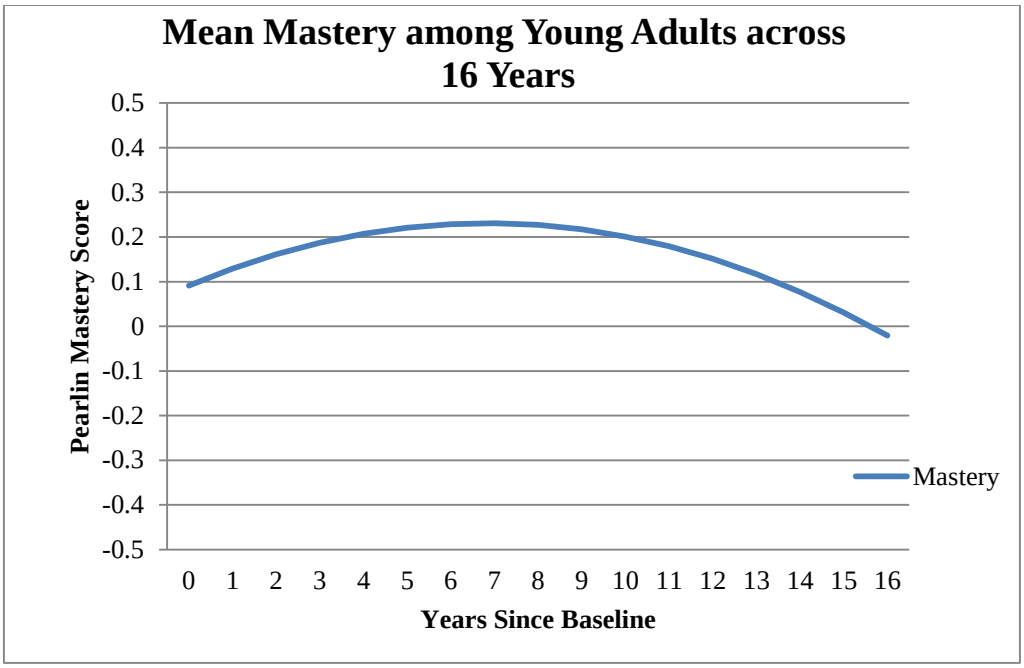

Figure 4.1 Mean Mastery among Young Adults across 16 years (Table 4.2, Model 1)

As outlined in table 4.2, the unconditional growth model (model 1) illustrates that mastery shares a linear relationship with time, in which mastery increases with time. However, a quadratic relationship was also significant, suggesting that mastery increases with the passage of time at a decreasing rate. Thus, mastery develops in a curvilinear manner, as depicted above in figure 4.1. For the quadratic model, intercept of .091 represents the average initial estimate of mastery for all participants at baseline. The significant mean associated with the first slope of .040 indicates that over time, there is an increasing trend of .040. A negative sign associated with the mean in the second slope (time squared) indicates that the general increase in mastery slows down over time. With each year, individual mastery increases at an average rate of .040 at a decreasing rate of .003.

Table 4.2 2 Level MLM on Mastery among Young Adults (N=1412)

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	.091* (.043)	.107* (.045)	.106* (.046)	.016 (.102)	-.044 (.108)
Time	.040*** (.011)	.040*** (.011)	.040*** (.012)	.040*** (.011)	.040*** (.011)
Time_Sq	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)
YngCI[a]		-.111 (.096)	-.105 (.114)	-.041 (.095)	-.040 (.095)
YngCI * Time			-.001 (.011)	---	---
Educ- <HS[b]				-.737* (.399)	-.737 (.399)
Educ- Some HS[c]				.020 (.144)	.034 (.143)
Educ- HS Grad[d]				-.131 (.088)	-.129 (.087)
Educ – Some College[e]				.039 (.084)	.050 (.084)
Married[f]				-.103 (.077)	-.106 (.077)
Income				5.93^{-6}** (2.03^{-6})	5.92^{-6}** ($.2.02^{-6}$)
Male[g]					.115 (.065)
Black [h]					.062 (.095)
OthRace[i]					.105 (.162)

Random Effects

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	.354*** (.047)	.351*** (.046)	.351*** (.046)	.330*** (.045)	.330*** (.045)
Intercept By Time	-.027** (.010)	-.026** (.010)	-.026** (.010)	-.028** (.010)	-.028** (.010)
Time	.013*** (.003)	.014*** (.003)	.013*** (.003)	.014*** (.003)	.014*** (.003)
Residual	.337*** (.020)	.336*** (.020)	.336*** (.020)	.334*** (.020)	.334*** (.020)

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5
-2LL	3507.0	3505.7	3505.7	3487.1	3483.0
AIC	3527.0	3527.7	3529.7	3521.1	3523.0
AICC	3527.2	3527.9	3529.9	3521.6	3523.6
BIC	3565.7	3570.2	3576.1	3586.8	3600.4
Chi Square	381.92***	382.69***	382.66***	376.44***	364.43***
ΔD	---	1.3	0	18.6**	4.1

***p<.001 ** p< .01 * p<.05

Note: raw estimates are presented first along with standard errors in parentheses.

[a] reference category is: healthy. [b-e] reference categories are: college graduate. [f] reference category is: unmarried. .[g] reference category is female. [h, i] reference categories are: white.

In table 4.2, model 1, I include estimates for an unconditional growth model of mastery among the young adult sample that supports the inclusion of the quadratic term (b=-.003, p<.001). Mastery is strongly and significantly associated with time. In model 2, I sought to answer the following research question: What is the relationship between early onset chronic illness and mastery? I examined the relationship between early onset chronic illness and mastery and found that on average, early onset illness is associated with lower mean mastery than remaining healthy (b=-.111), but, this finding was not statistically significant. Thus, there is no evidence that a disparity in mastery exists between young adults who experience early onset illness and those without early onset chronic illness. This non-significant finding likely reflects that within each subsample, cases with the lowest levels of mastery attritted from the study, were subsequently deleted from the sample, and are not represented in these analyses (For a full discussion of sample selection and missing data, review chapter 3). Consequently, adults with early onset illness and their healthy same age peers possess similarly high levels of mastery. Results presented are conservative estimates and should be interpreted as such. Hypothesis 1a stated that early onset chronic illness is associated with lower mastery compared to those without early onset chronic illness. This hypothesis was not supported.

In model 3, I included an interaction of the linear term and early onset illness to assess if adults with early onset illness and the healthy differ in their rate of change in mastery. This interaction was non-significant (b=-.001), meaning these two groups of young adults acquire mastery at a similar rate across the study period. As a result, this variable was not included in the subsequent models. The main effect of early onset illness remained non-significant and effect size decreased. Linear and quadratic effects remained significant (p<.001).

Next, I controlled for the effects of socioeconomic characteristics, including marital status, educational attainment, and annual income, in model 4. Of these variables, education and income shared a significant relationship with mean mastery. Specifically, completion of less than 9th grade predicted .737 standard points lower on the mastery scale than attainment of a college degree ($p<.05$). A one unit increase ($10k) in income was associated with 5.93^{-6} standard points higher on the Pearlin Mastery Scale ($p<.01$). Early chronic illness remained a non-significant predictor of mastery (b=-.041) and decreased considerable in effect size from -.105 to -.041.

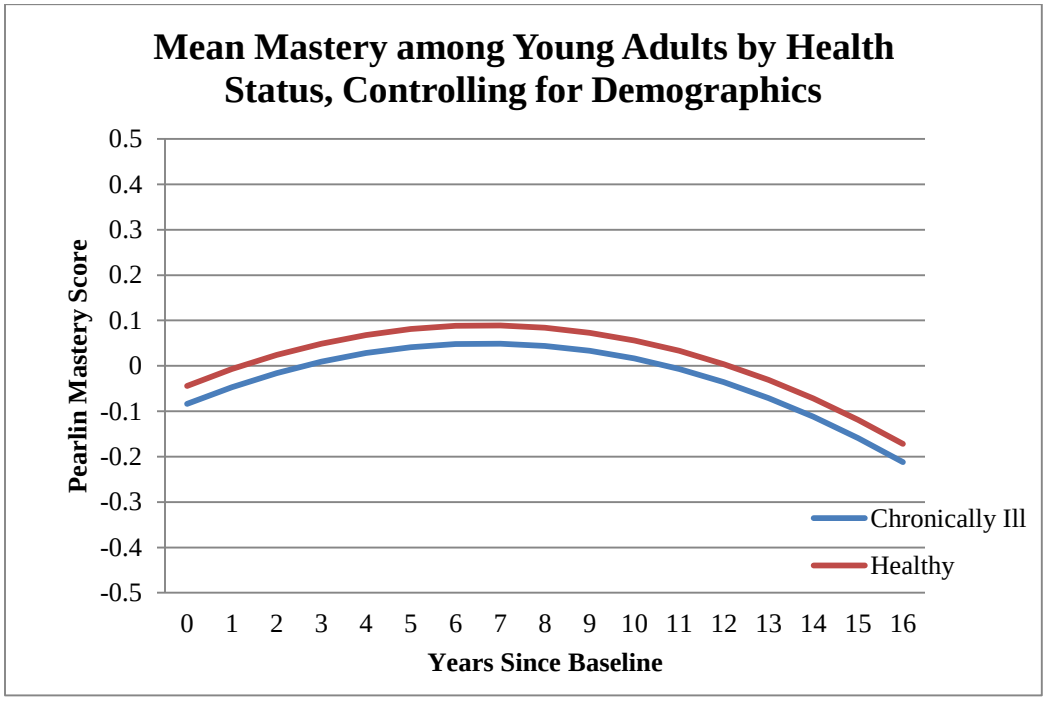

Figure 4.2 Mean Mastery among Young Adults by Health Status, Controlling for Demographics (Table 4.2, Model 5)

In model 5, I included additional demographic characteristics, sex and race (figure 4.2). Among young people, men and women did not significantly differ on mean mastery. Being black or another "nonwhite/nonblack" race was also non-significant. In review, hypothesis 1a stated that early onset chronic illness is associated with less mastery compared to those without early onset chronic illness. The results demonstrate that becoming ill during young adulthood (24-35)

is not associated with lower mean mastery than remaining healthy. As a result, hypothesis 1a was not supported.

4.3.2 Random Effects

In order to understand if average change in mastery differs by individuals, I estimated random effects and allowed intercept and slope to vary by person. I found that mastery at baseline, represented by the intercept, and rate of change in mastery, represented by the slope, significantly vary between people. In models 1-5, the statistically significant covariance between the slopes and intercepts suggests that as intercepts increase, slopes decrease. For example, in model 5, a one unit increase in baseline mastery is associated with a .028 decrease in rate of change in mastery. Thus, mastery is negatively associated with rate of change. The greater individual mastery is at baseline, the more gradual the rate of intraindividual change in mastery across the 16 year study period. Across all models, residual estimates decrease very slightly (approx. 33.7% to 33.4%), suggesting that predictors not included in analyses explain intraindividual and interindividual change in mastery more fully. After running tests of nested models, model 4, which included health status and measures of socioeconomic status, was determined to be the best fitting model.

4.3.3 Summary

1. Among this sample, early onset chronic illness is not associated with lower mean mastery than remaining healthy, even when controlling for socioeconomic and demographic characteristics.

2. Young adults with greater mastery at baseline experience more gradual change in their trajectories than do those with less mastery at study baseline.

In the section below, I present results for the examination of the effects of timing of illness onset on mastery. In these analyses, the sample is limited to cases between the ages of 24 and 96 years of age that were healthy at baseline, but subsequently reported a chronic illness at

one or more waves. This sample is referred to as the "All Ages Restricted to Chronically Ill" sample.

4.4 Results: Effects of Timing of Illness Onset on Mastery

Table 4.3 Model Fit Statistics on Mastery among the All Ages Restricted to Chronically Ill Sample (N=2148)

(N=2148)	Model 1	Model 2	Model 3	Model 4
	Mastery	Mastery, Time (fixed)	Mastery, Time (fixed & random)	Mastery, Time (fixed & random), Time_sq (fixed and random)
AIC	5964.8	5941.5	5840.7	5743.5
AICC	5964.8	5941.6	5840.7	5763.5
BIC	5977.7	5958.7	5866.4	5763.6
-2LL	5958.8	5933.5	5828.7	5806.4
Chi Square	381.72***	390.55***	495.43***	556.62***
ΔD	---	25.33**	74.8**	22.3**

***p<.001 ** p< .01 * p<.05

First, I fitted an "empty model" to estimate how much of the variation in mastery during adulthood occurs between-subjects. The intra-class correlation (ICC) of .3299 suggests that approximately 32.99 % of variation in mastery is attributable to between-person factors. Next, I estimated mastery trajectories by including a linear term (time) as a fixed effect only in model 2 and as fixed and random effects in model 3. Lastly, I included a quadratic term as fixed and random effects into model 4, allowing for random intercept and slope. In assessing model fit, -2 log likelihood, AIC, and BIC values were compared in deciding if model fit was significantly improved by inclusion of the quadratic term (time squared). Nested models tests indicated that model fit was improved by the inclusion of additional terms in each subsequent and "fuller" model. Change in deviance (ΔD) was significant at the .01 level from model 1 to model 2, model 2 to model 3, and model 3 to model 4. As evidenced in table 4.3, the quadratic form best fit the data and appear in the results table (table 4.4) as the first model.

Table 4.4 Level MLM on Mastery among the All Ages Restricted to Chronically Ill Sample (N=2148)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept	.071 (.040)	.050 (.043)	-.016 (.117)	-.022 (.123)	-.059 (.125)	-.387 (.209)
Time	.044*** (.010)	.044*** (.001)	.047*** (.014)	.044*** (.010)	.044*** (.010)	.045*** (.010)
Time_Sq	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)
MidCI[a]		-.044 (.099)	.063 (.123)	-.021 (.098)	-.013 (.097)	-.005 (.097)
LateCI [b]		.192 (.012)	.240 (.147)	.166 (.115)	.159 (.114)	.173 (.114)
MidCI*Time			-.003 (.011)	---	---	---
LateCI*Time			-.007 (.013)	---	---	---
Educ- <HS[c]				-.343 (.176)	-.353* (176)	-.324 (.176)
Educ- Some HS [d]				-.096 (.111)	-.083 (.110)	-.071 (.110)
Educ- HS Grad[e]				-.144 (.078)	-.140 (.077)	-.134 (.077)
Educ – Some College[f]				-.012 (.078)	-.012 (.078)	-.010 (.078)
Married[g]				-.067 (.065)	-.096 (.065)	-.097 (.065)
Income				6.089^{-6}*** (1.609^{-6})	5.987^{-6}*** (1.599^{-6})	5.922^{-6}*** (1.595^{-6})
Male[h]					.149** (.058)	.145* (.058)
Black [i]					-.008 (.084)	-.001 (.084)
OthRace[j]					-.269 (.172)	-.260 (.172)
Function						.080 (.041)
Comorbid						-.008 (.028)

Random Effects

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept	.455*** (.049)	.452*** (.048)	.452*** (.048)	.432*** (.047)	.424*** (.047)	.423*** (.046)
Intercept By Time	-.034*** (.010)	-.034*** (.010)	-.034** (.010)	-.035** (.010)	-.035*** (.010)	-.034*** (.010)
Time	.014*** (.003)	.014*** (.003)	.013*** (.003)	.014*** (.003)	.014*** (.003)	.014*** (.003)
Residual	.361*** (.017)	.360*** (.017)	.360*** (.017)	.358*** (.017)	.359*** (.017)	.358*** (.017)

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
-2LL	5743.5	5739.4	5739.0	5708.3	5699.4	5695.2
AIC	5763.5	5763.4	5767.0	5744.3	5741.4	5741.2

AICC	5763.6	5763.5	5767.2	5744.6	5741.8	5741.7
BIC	5806.4	5814.8	5827.0	5821.4	5831.4	5839.7
Chi Square	556.62***	555.45***	555.75***	546.44***	511.64***	508.53***
ΔD	---	4.1	0.4	30.7**	8.9*	4.2

***p<.001 ** p< .01 * p<.05
Note: raw estimates are presented first along with standard errors in parentheses.
[a-b] reference category is: early onset chronic illness. [c-f] reference categories are: college graduate. [g] reference category is: unmarried. [h] reference category is: female. [i-j] reference categories are: white.

4.4.1 Fixed Effects

Table 4.4 presents six nested models estimating continuous mastery among the all ages restricted to chronically ill sample. Fixed effects are presented first in the table, followed by random effects and subsequently, model fit statistics. Tests of nested models (ΔD) revealed that model 5 is the best fitting, as the inclusion of SES and demographic characteristics significantly improved model fit (p<.01). In model 1, I include estimates for an unconditional growth model of mastery that supports the inclusion of the quadratic term (b=-.003, p<.001). Mastery is strongly and significantly associated with time (figure 4.3). As depicted, among chronically ill adults between 24 and 96 years of age, change in mastery among chronically ill adults of all ages is curvilinear and convex. Mastery increases across the first half of the study period and begins a decline around year 8 of the study. By year 16, wave 4 of data collection, average growth has slowed and returned to baseline levels.

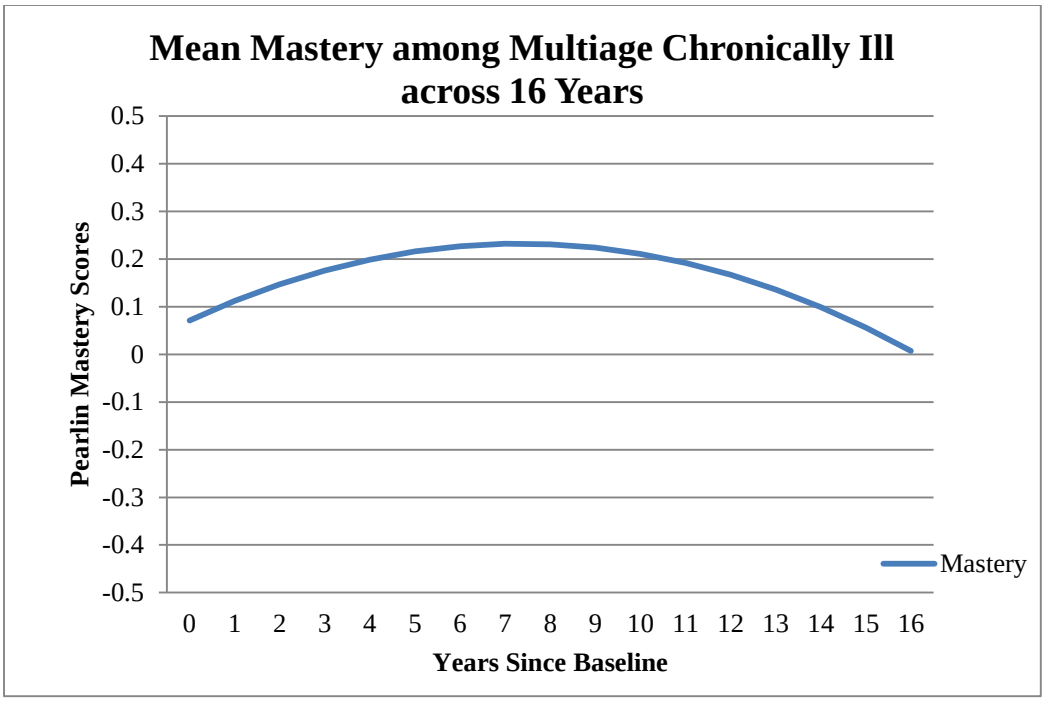

Figure 4.3 Mean Mastery among Multiage Chronically Ill across 16 Years (Table 4.4, Model 1)

In model 2, I included timing of illness onset into the model in order to test hypothesis 3a. Hypothesis 3a states that early onset chronic illness is associated with lower mastery than illness onset at mid-life or late-life. There were no statistically significant findings to suggest that differences in mean mastery exist between individuals who become ill as young, middle or older adults. Similarly, no statistically significant differences in linear rate of change between adults with early, mid-life, or late-life illness onset were detected. However, the linear and quadratic terms remained significant ($p<.001$) and as depicted in figure 4.4, each of the subsamples follow similar trajectories of growth in mastery with initial values above the mean, increases that level off at the study midpoint (years 8 -9), and subsequent declines that reapproach their respective baseline values.

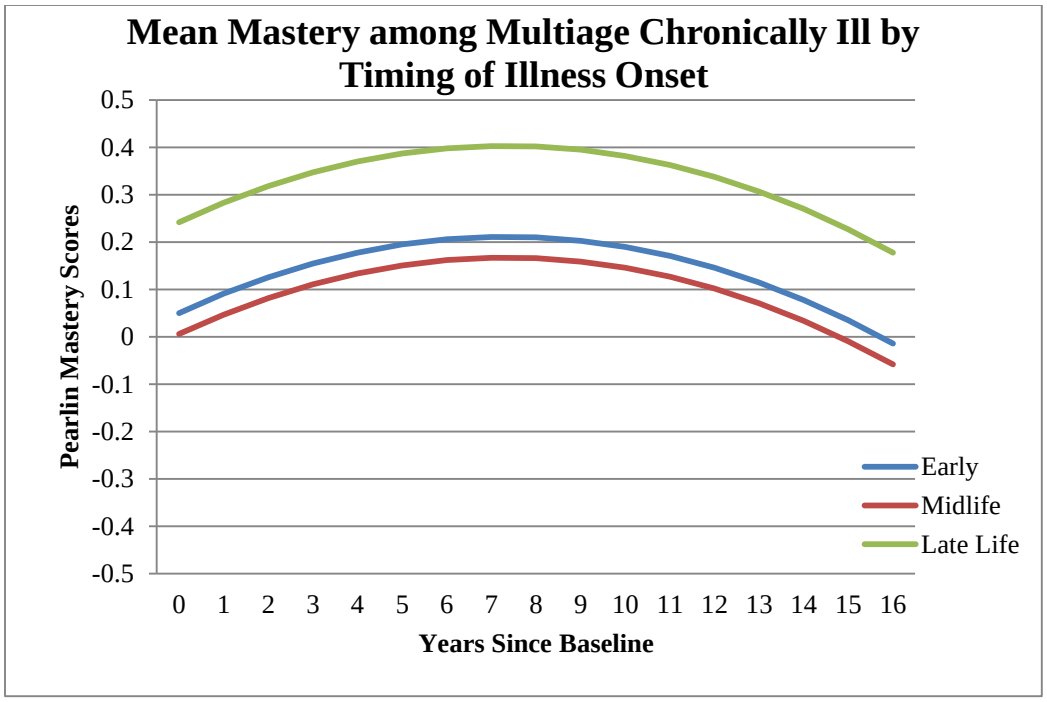

Figure 4.4 Mean Mastery among Multiage Chronically Ill by Timing of Illness Onset (Table 4.4, Model 2)

Next, I included interactions of timing of illness onset variables and time in order to assess if subgroups of chronically ill people acquire or lose mastery at different rates. Both interactions were non-significant, affirming that timing of illness onset is not predictive of rate of change in mastery, among this sample. These interactions were not included in subsequent models. The main effects, mid-life onset (b=.063) and late-life onset (b=.240), remained statistically non-significant, but increased in effect size.

In model 4, I controlled for the effects of socioeconomic characteristics (educational attainment, marital status, and income) and found that when controlling for SES, timing of illness onset remains non-significant. Educational attainment and marital status were also non-significant. Annual income was significantly associated with mastery, as a one standard unit ($10k) increase in income was associated with a 6.089^{-6} increase in mastery. After inclusion of the SES measures, the effect sizes of mid-life and late-life onset decreased dramatically, illustrating that income partially explains any effect timing of illness may have on mastery.

I included sex and race into model 5. Sex was statistically significant ($p<.01$). On average, men report higher mastery than women ($b=.149$). Race was a non-significant predictor of mastery. After controlling for sex and race, income remained statistically significant ($b=5.987^{-6}$, $p<.001$) and education below the 9th grade became statistically significant ($p<.05$). Chronically ill adults with less than a 9th grade education possess mastery that is .353 points lower than chronically ill adults with a college degree. Timing of illness onset remained non-significant and main effect sizes decreased again. A graphic representation of the effects of timing of illness onset on mastery, controlling for socioeconomic and demographic characteristics (model 5) is included below as figure 4.5. This figure clearly illustrates the lack of a statistically difference in mastery trajectories among adults with early and mid-life onset, as each group's trend lines are nearly indistinguishable from one another. Both of these groups begin the study very slightly below mean levels of mastery, increase and hover very slightly (0.1) above the mean, level off, and ultimately return to levels of mastery that are slightly below average (-0.1). Alternatively, adults with late-life onset begin the study with higher than average mastery and retain these above average levels across the 16 year period.

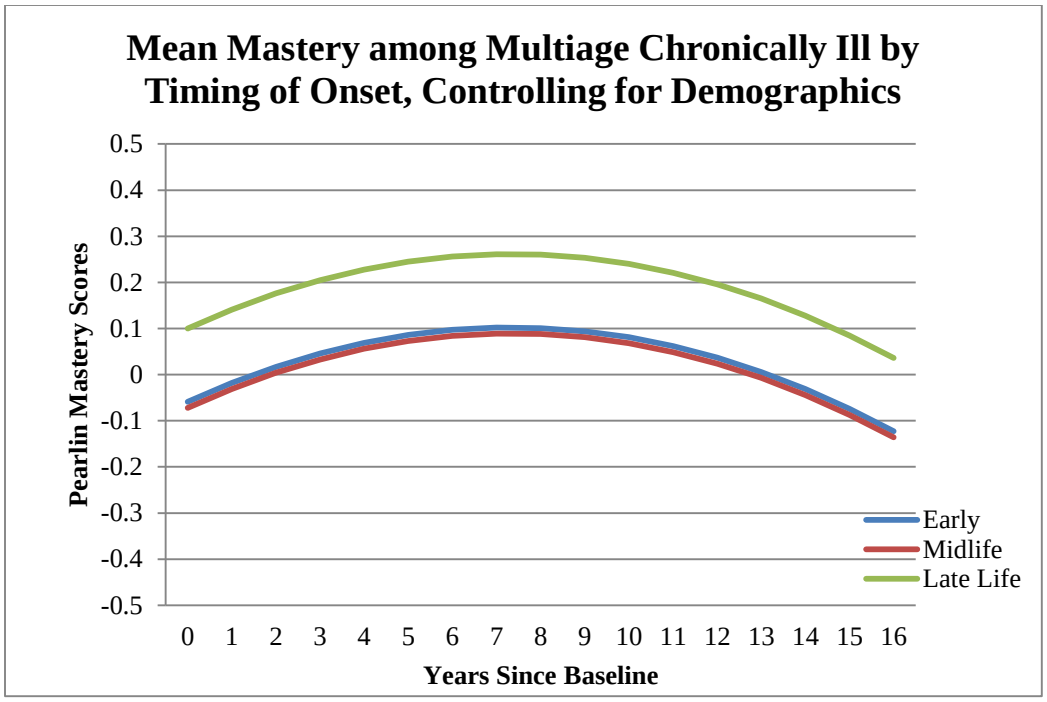

Figure 4.5 Mean Mastery among Multiage Chronically Ill by Timing of Onset, Controlling for Demographics (Table 4.4, Model 5)

Lastly, I included the illness characteristics, number of comorbid chronic health conditions and functional impairment, in model 6 to control for their effects on mastery among chronically ill adults. Neither functional impairment nor comorbidity was significantly associated with mastery among this sample. However, their inclusion into the model reduced the effect size of mid-life onset from -.013 to -.005 and increased the effect size of late-life onset from .159 to .173. As the estimates and illustration (figure 4.6) suggest, controlling for illness characteristics makes mastery trajectories of adults with early and mid-life onset more similar. As depicted in the figure, the inclusion of functional impairment and comorbidity shifts all three trajectories downward below the mean. Even adults with late-life onset who previously were thought to retain higher than average mastery across the study period report mastery that is .2 standard points lower than average at baseline, after controlling for the effects of illness characteristics.

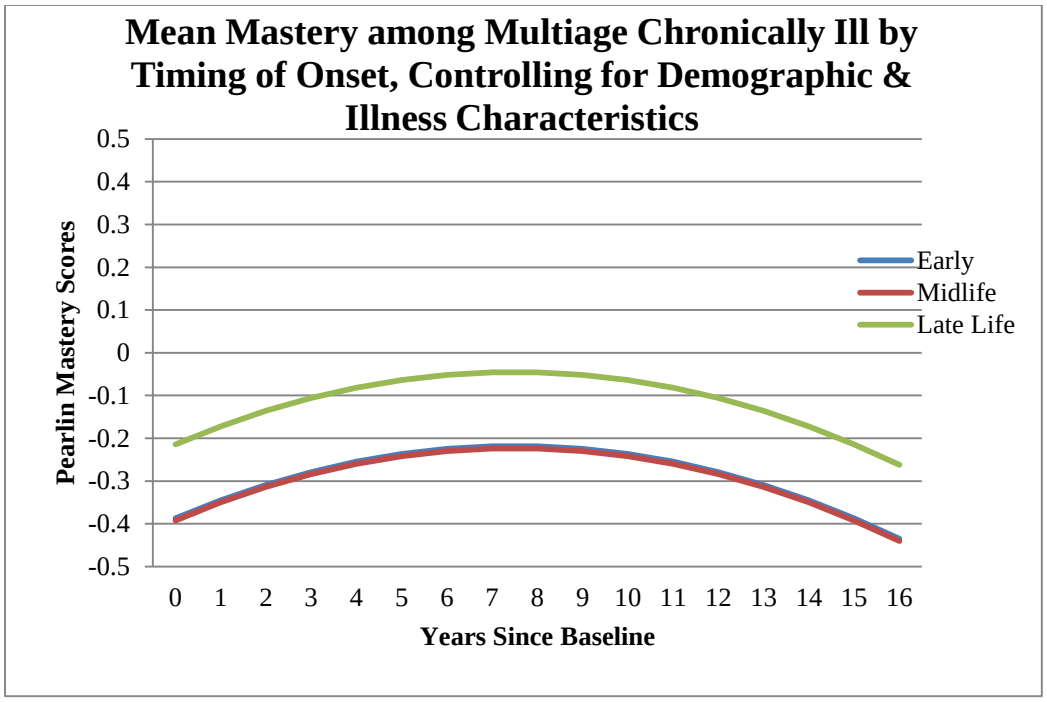

Figure 4.6 Mean Mastery among Multiage Chronically Ill by Timing of Onset, Controlling for Demographic and Illness Characteristics (Table 4.4, Model 6)

This finding that mean mastery is not significantly associated with chronic illness at any stage of the adult life course counters the prevailing knowledge in this area. Based on the literature, timing of chronic illness onset and mastery should share a significant association. Although in these models, I controlled for factors known to influence mastery, such as demographic factors and influential illness characteristics, functional impairment and comorbidity, the chronic illness – mastery relationship remained non-significant for those with early, midlife, and late life onset. These findings suggests that when considering age at chronic illness onset, a variable that has not yet been identified is likely suppressing this expected and important relationship.

4.4.2 Random Effects

Estimates for random effects illustrate that among chronically ill adults, significant heterogeneity in mastery at baseline and growth in mastery across 16 years exist. Random

intercept, representative of baseline mastery, is negatively associated with rate of change in mastery. Chronically ill adults with greater mastery at baseline experience more gradual change in their trajectories than do those with less mastery at study baseline. On average, individuals' rate of change in mastery varies by .013 -.014 units ($p<.001$). Thus, estimates confirm that significant variation in trajectory of mastery and mastery at baseline occurs within and between people. Across models, approximately 36% of variation in depressive symptoms is accounted for by between-persons factors ($p<.001$).

4.4.3 Summary

In review, results from analyses presented in table 4.4 confirm the following:

1. Mid-life onset and late-life onset are not significantly associated with higher mastery than onset during young adulthood (24-35), even when controlling for socioeconomic and demographic characteristics.

2. Timing of illness onset is not predictive of rate of change in mastery across the 16 year study period.

3. Chronically ill adults with greater mastery at baseline experience more gradual change in their trajectories than do those with less mastery at study baseline.

In the chapter that follows, I extend analyses to the examination of the effects of timing of chronic illness onset on depressive symptoms.

5 RESULTS: DEPRESSIVE SYMPTOMS

5.1 Analyses: Depressive Symptoms as Outcome

In analyses detailed below, I fitted two-level random intercepts and slopes models using maximum likelihood estimation, in which I allowed both intercepts and slopes to vary across individuals. In the case of change in a measure of mental health, depressive symptoms, it was most reasonable to assume that individuals differ in their level of depressive symptoms at baseline and rate of change across a 16 year period. Thus, a random intercepts and slopes model was most appropriate for examining the primary research questions of this study.

In all analyses, I model the effects of 1) early onset chronic illness and 2) timing of chronic illness onset on depressive symptoms across 4 time points (16 years). Time was measured to reflect the number of years since baseline interview in 1986. Consequently, time was measured as 0 (1986), 3 (1989), 8 (1994), and 16 (2002). Time also serves as a measurement proxy of age. Excluding respondent race, income, marital status, educational attainment and sex, all predictors were entered into the models as time varying.

After assessing multiple covariance structures, I decided that an unstructured covariance structure was most appropriate, as baseline estimates of depressive symptoms and their respective trajectories are strongly correlated. Additionally, an unstructured covariance structure allowed for random effects for depressive symptoms at baseline (intercepts) and time (slopes), and the interaction between intercepts and slopes (Singer and Willett 2003; 257 – 260). Below, I present this study's research questions and related hypotheses and proceed to discussion of results.

5.2 Research Questions and Hypotheses:

Research Question #2: What is the relationship between early onset chronic illness and depressive symptoms? Does mastery mediate the relationship between early onset chronic illness and depressive symptoms?

Hypothesis 2a: Early onset chronic illness is significantly associated with higher depressive symptoms compared to those without early onset chronic illness.

Hypothesis 2b: Mastery mediates the relationship between early onset chronic illness and depressive symptoms.

Research Question #4: Is early onset chronic illness (24-35) associated with greater depressive symptoms than illness onset at the more socially normative life stages of mid-life (36-64) and late-life (65 years and older)? Does mastery mediate or moderate this relationship?

Hypothesis 4a: Early onset chronic illness is associated with greater depressive symptoms than illness onset at mid-life or late-life.

Hypothesis 4b: Mastery mediates the relationship between timing of illness onset and depressive symptoms.

Hypothesis 4c: Mastery moderates the relationship between timing of illness onset and depressive symptoms.

5.3 Results: Effect of Early Onset Chronic Illness on Depressive Symptoms

Prior to running analyses, I fit an "empty model" to estimate the intra-class correlation (ICC) for depressive symptoms among the young adult sample. The ICC was estimated as .3799, meaning between-person factors explain 37.99 % of variance in depressive symptoms among these young adults. In table 5.2, model 1, the statistically significant coefficients ($p<.001$) for the linear and quadratic terms suggest that depressive symptoms are associated with the passage of time. Specifically, with each year depressive symptoms decrease at an average rate of -.067 at an increasing rate of .003. Tests of nested models indicate that model 4, which includes time and time squared as fixed and random effects, best fit the data. Each "fuller" model was a significant ($p<.01$) improvement upon its preceding nested model.

Table 5.1 Model Fit Statistics on CESD among Young Adults (N=1412)

(N=1412)	Model 1	Model 2	Model 3	Model 4
	CESD	CESD, Time (fixed)	CESD, Time (fixed & random)	CESD, Time (fixed & random), Time_sq (fixed and random)
AIC	3624.6	3587.6	3579.3	3524.4
AICC	3624.6	3587.6	3579.4	3544.4
BIC	3636.2	3603.1	3602.5	3544.6
-2LL	3618.6	3579.6	3567.3	3583.1
Chi Square	327.93***	344.41***	356.68***	388.62***
ΔD	---	39.0**	12.3**	15.8**

***p<.001 ** p< .01 * p<.05

As illustrated in figure 5.1, the form of growth in depressive symptoms among young adults is best characterized as curvilinear and takes a U shape. After baseline, depressive symptoms decrease at an increasing rate and level off around year 11 of the study period at approximately -0.4 standard points. At this point in the study, young adults range in age from 35 to 46 years of age, with the overwhelming majority having entered mid-life. As depicted, after this point CESD scores increase very slightly on average.

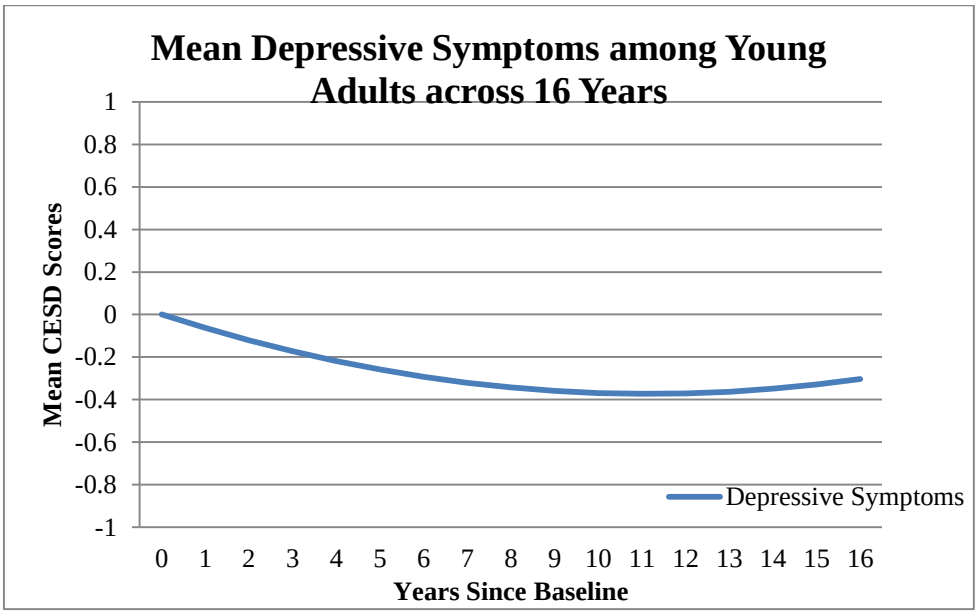

Figure 5.1 Mean Depressive Symptoms among Young Adults across 16 Years (Table 5.2, Model 1)

Table 5.2 2 Level MLM on CESD among Young Adults (N=1412)

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	.001 (.049)	-.038 (.051)	-.044 (.052)	-.006 (.046)	.143 (.099)
Time	-.067*** (.012)	-.067*** (.012)	-.066*** (.012)	-.054*** (.012)	-.053*** (.012)
Time_Sq	.003*** (.001)	.003*** (.001)	.003*** (.001)	.002** (.001)	.002** (.001)
YngCI[a]		.276** (.103)	.325** (.122)	.242** (.087)	.191* (.085)
YngCI*Time			-.007 (.009)	---	---
Mastery				-.335*** (.025)	-.333*** (.025)
Educ- <HS[b]					.178 (.365)
Educ- Some HS[c]					.100 (.128)
Educ- HS Grad[d]					.133 (.077)
Educ – Some College[e]					.040 (.075)
Married[f]					-.114 (.069)
Income					4.27^{-6} (1.81^{-6})
Male[g]					-.069 (.058)
Black[h]					.194* (.088)
OthRace[i]					.333* (.143)

Random Effects

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	.497*** (.062)	.489*** (.061)	.490*** (.062)	.375*** (.052)	.343*** (.049)
Intercept By Time	-.046*** (.012)	-.046*** (.012)	-.046*** (.012)	-.047*** (.011)	-.045*** (.011)
Time	.014*** (.003)	.014*** (.003)	.014*** (.003)	.014*** (.003)	.014*** (.003)
Residual	.378*** (.022)	.377*** (.022)	.377*** (.022)	.357*** (.021)	.355*** (.021)

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5
-2LL	3524.4	3517.3	3516.7	3360.7	3329.3
AIC	3544.4	3539.3	3540.7	3384.7	3371.3
AICC	3544.6	3539.4	3540.9	3384.9	3371.9
BIC	3583.1	3581.8	3587.1	3431.1	3452.5
Chi Square	388.62***	384.24***	384.55***	210.45***	189.92***
ΔD	---	7.1**	0.6	156.0**	31.4**

***p<.001 ** p< .01 * p<.05

Note: raw estimates are presented first along with standard errors in parentheses.

[a] reference category is: healthy. [b-e] reference categories are: college graduate. [f] reference category is: unmarried. [g] reference category is female. [h, i] reference categories are: white.

5.3.1 Fixed Effects

Table 5.2 presents results of analyses of the effect of early chronic illness onset on depressive symptoms. These analyses were performed on the previously defined "young adult sample," which consists of young adults with early onset chronic illness and peers who remained healthy for the study's duration. Model 1 includes estimates for linear and quadratic terms and fixed and random effects. Tests of nested models (ΔD) and comparison of AIC and BIC values indicate that model 5, which includes health status, socioeconomic, and demographic characteristics as predictors, is the best fitting model. Change in -2LL reveals that models were significantly improved from model 1 to model 2, model 3 to model 4, and model 4 to model 5.

In model 2, I included early chronic illness onset as a predictor and found a statistically significant association with depressive symptoms. Young adults who experience early onset illness experience greater depressive symptoms at baseline than those without early onset chronic illness. Young adults with chronic illness report depressive symptoms .276 units higher than healthy same age peers. Hypothesis 2a stated that early onset chronic illness is significantly associated with higher depressive symptoms compared to those without early onset chronic illness. As a result, hypothesis 2a is supported by these findings because mean depressive symptoms differ by health status.

In model 3, I included an interaction between early onset illness and time in order to examine if rate of change differs by health status. The interaction between early onset illness and the linear term was non-significant (b=-.007), meaning that linear and quadratic growth in depressive symptoms do not differ between adults with early onset chronic illness and the healthy. This interaction was not included in subsequent models. Early onset illness remained

significant ($p<.01$), with young adults with chronic illness reporting depressive symptoms .325 units higher than healthy same age peers.

Due to mastery's noted function as a mediator between stressors and outcomes, I entered mastery as a time-varying predictor into model 4 and examined if it mediates the relationship between early onset chronic illness and depressive symptoms. In this model, the main effect of early onset illness remained statistically significant ($b=.242$, $p<.01$), although the effect size decreased, suggesting that mastery does have an effect on the relationship between early illness onset and depressive symptoms at baseline.

Mastery is negatively associated with mean depressive symptoms ($b=-.335$, $p<.001$) and its inclusion in the model decreased effect size for the bivariate relationship between early onset illness and depressive symptoms (model 3, $b=.325$ to model 4, $b=.242$). To review, hypothesis 2b stated that mastery mediates the relationship between early onset chronic illness and depressive symptoms. Findings from chapter 4 that early onset chronic illness was not significantly associated with mastery suggests that mastery cannot mediate the early onset chronic illness – depressive symptoms relationship. Thus, hypothesis 2b was not supported because a significant bivariate relationship between early onset illness and mastery was not detected.

Lastly, I included socioeconomic and demographic characteristics into model 5. Of these characteristics, only race was statistically significant. Being black ($b=.194$, $p<.05$) or another "non-white" race ($b=.333$, $p<.05$) was associated with higher levels of depressive symptoms. Among young adults, education, marital status, and income were not statistically significant predictors of depressive symptoms. The main effect of early onset illness decreased in effect size

(b=.191) and significance, but remained significant (p<.05). Estimate of linear (b=.053) and quadratic (b=-.002) rate of change remained significant (p<.05).

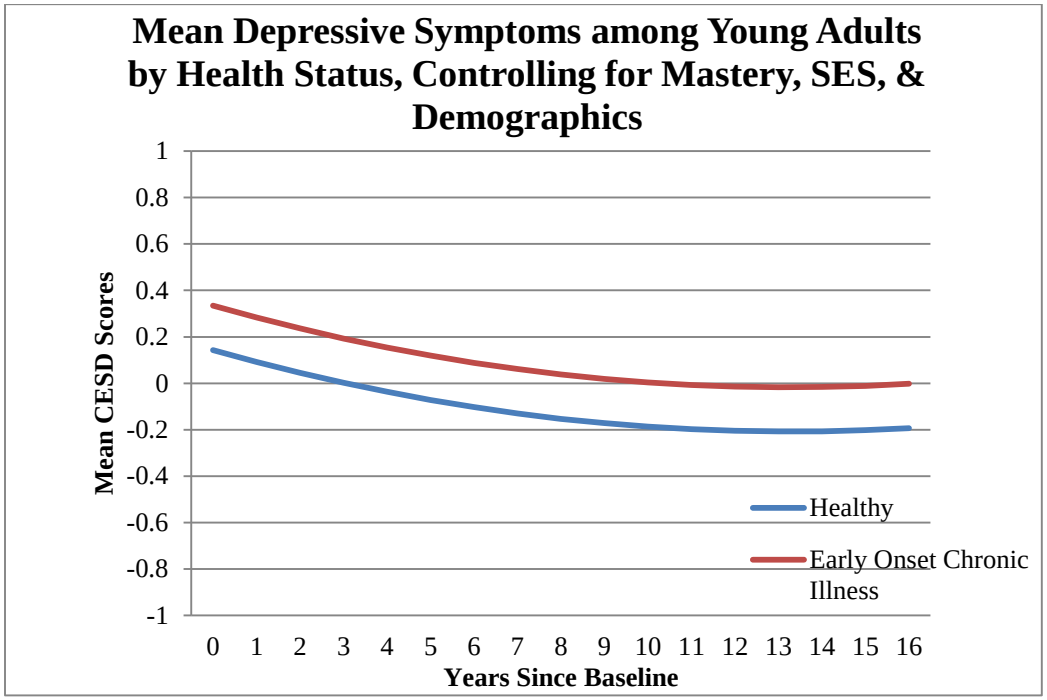

Figure 5.2 Mean Depressive Symptoms among Young Adults by Health Status, Controlling for Mastery, SES & Demographics (Table 5.2, Model 5)

Figure 5.2 illustrates trajectories of depressive symptoms by health status after controlling for mastery, SES, and demographic characteristics. Interestingly, young adults who later experience early onset chronic illness report higher depressive symptoms at baseline than peers who remain healthy across the study period. This differential (≈.2 standard points) remains consistent across the 16 years study period. Depressive symptoms among both groups of young adults level off around year 10 and remain mostly flat for the last 6 years of the study period.

5.3.2 Random Effects

By examining estimates of the random effects, it is clear that significant variation exists within the young adult sample in depressive symptoms at baseline and rate of intraindividual growth of depressive symptoms (table 5.2). Random effects provide information on average deviation in depressive symptoms for each subject from the overall mean. In all models, intercept

was negatively associated with slope, which means that higher depressive score at baseline predicts slower intraindividual growth. Approximately one third of variance in depressive symptoms is explained by within-person factors that are unobserved and thus, unaccounted for in the analytic models. In each of the models, there remains considerable unexplained variation in depressive symptoms, as the residual ranges from .378 in model 1 to - .355 in model 5.

5.3.3 Summary

In review, results from analyses presented in table 5.2 confirm the following:

1. Adults who later develop early onset chronic illness report higher levels of depressive symptoms prior to illness onset than do peers who remain healthy.

2. There is no statistical difference in the rate of change in depressive symptoms among adults with early onset illness and healthy peers.

3. Mastery does not mediate the effect of early onset chronic illness and depressive symptoms.

4. Controlling for education, marital status, annual income, and race, early onset chronic illness remains a significant predictor of depressive symptoms, with ill adults reporting higher levels of depressive symptoms than the healthy.

5. Among young adults, greater depressive symptoms prior to the experience of illness onset (e.g. baseline) are associated with more gradual intraindividual growth in depressive symptoms.

6. Individual variation in depressive symptoms prior to illness onset (e.g. baseline) partially explains interindividual and intraindividual variation in growth of depressive symptoms.

Below, I present the results for analyses of the timing of illness onset on depressive symptoms. The analyses below were conducted using the previously defined "All Ages Restricted to Chronically Ill Sample."

5.4 Results: Effects of Timing of Illness Onset on Depressive Symptoms

Initially, I fit an "empty model" to estimate the intra-class correlation (ICC) for depressive symptoms among the all ages restricted to chronically ill sample. The ICC was

estimated as .4479, meaning between-person factors explain 44.79 % of variance in depressive symptoms among chronically ill adults. Table 5.3 includes information pertaining to model fit and outlines how I arrived at the conclusion that growth in depressive symptoms among the all ages restricted to chronically ill sample is best modeled with a linear and quadratic term. In model 4, model fit is significantly improved by the inclusion of the quadratic term (time*time). Tests of nested models (ΔD) indicated that model 4, which includes time and time squared as fixed and random effects, was the best fitting model to the data. Across models 1 through 4, each "fuller" and subsequent model was better fitting ($p<.01$) than the model nested within it.

Table 5.3 Model Fit Statistics on CESD among the All Ages Restricted to Chronically Ill Sample (N=2148)

(N=2148)	Model 1	Model 2	Model 3	Model 4
	CESD	CESD, Time (fixed)	CESD, Time (fixed & random)	CESD, Time (fixed & random), Time_sq (fixed and random)
AIC	5568.8	5554.4	5501.0	5436.4
AICC	5568.8	5554.4	5501.0	5436.5
BIC	5581.7	5571.5	5526.7	5479.2
-2LL	5562.8	5546.4	5489.0	5416.4
Chi Square	587.79***	595.25***	652.66***	713.98***
ΔD	---	16.4**	57.4**	72.6**

***p<.001 ** p< .01 * p<.05

Among adults with chronic illness, depressive symptoms follow a curvilinear pattern with an initial decline, leveling off, and eventual increase (figure 5.3). Change is very gradual, with standard CESD scores remaining between the mean and 0.4 standard points below the mean for the duration of the study period. As a result of including the linear and quadratic terms in the models, growth in depressive symptoms was controlled for prior to the introduction of any predictor or control variables.

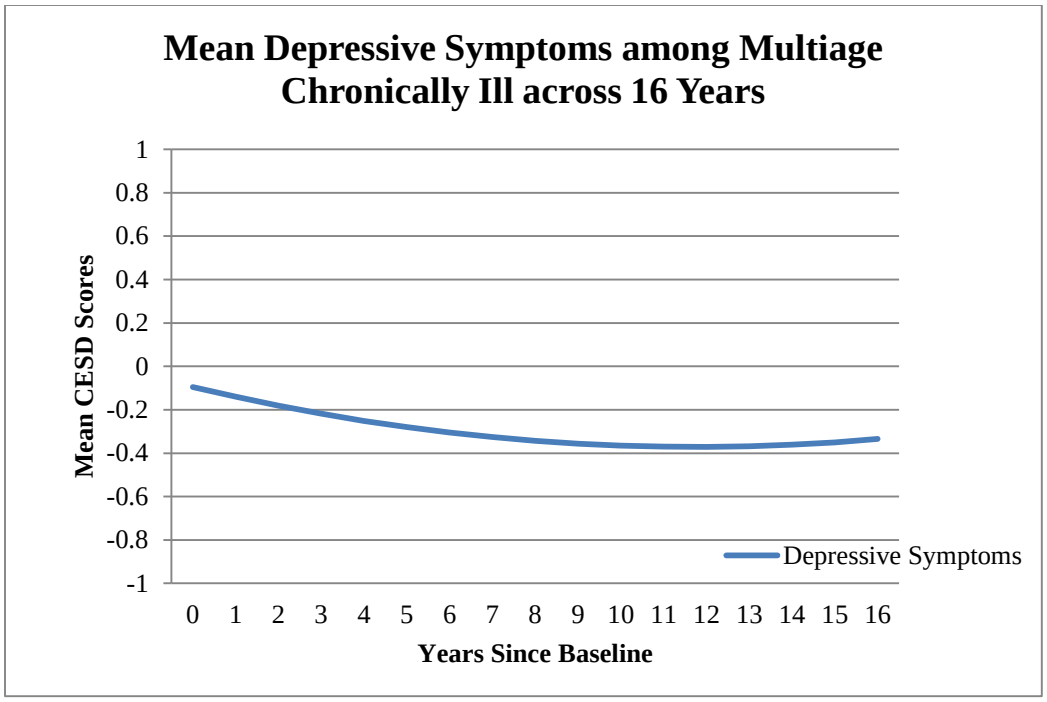

Figure 5.3 Mean Depressive Symptoms among Multiage Chronically Ill across 16 Years (Table 5.4, Model 1)

5.4.1 Fixed Effects

Progressing to analyses, I fitted a two level hierarchal model that included random and fixed effects (table 5.4). I conducted tests of nested models, which compared change in -2LL from a simpler, nested model to a fuller model that included additional predictors. Across models 1 through 8, model fit was significantly improved ($p<.05$) by the inclusion of additional predictors. Based off of these tests and comparisons of AIC and BIC estimates, model 8, which includes timing of illness onset, mastery, socioeconomic, demographic, and illness characteristics, and interactions of timing and mastery, was the best fitting and most complete model.

In table 5.4, model 1 includes estimates of the intercept, time (the linear effect), and time squared (the quadratic effect). Each estimate was significant. In model 2, I tested hypothesis 4a that states that early onset chronic illness (24-35) is associated with greater depressive symptoms than mid-life or late-life onset. I entered two dummy variables, mid-life onset and late-life

onset[21], and found that mean depressive symptoms significantly differ between adults with late-life onset and those with early onset (b=-.452, p<.001). Similarly, onset at mid-life is associated with mean CESD scores that are .242 lower than adults with early onset (p<.05). Interestingly, these differences in mean depressive symptoms were present at baseline, prior to any respondents becoming ill. Since all subjects were healthy at baseline, these group differences in depressive symptoms at baseline likely reflect developmental differences that are not linked to the chronic illness experience. However, this age group variation in "starting point" is important in considering how the disruption of illness may contribute to further disparities at a later time.

[21] Early onset illness, illness that occurred between the ages of 24 and 35 years of age, served as the reference to the two other categories.

Table 5.4 2 Level MLM on CESD among the All Ages Restricted to Chronically Ill Sample (N=2148)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Intercept	-.095* (.041)	0.161 (.101)	.261* (.118)	.253* (.105)	.300* (.124)	.255* (.126)	1.449*** (.186)	1.456*** (.185)
Time	-.047*** (.010)	-.047*** (.010)	-.060*** (.013)	-.046*** (.012)	-.045*** (.012)	-.045*** (.012)	-.048*** (.012)	-.049*** (.012)
Time_Sq	.002*** (.001)	.002*** (.001)	.002*** (.001)	.001* (.001)	.001* (.001)	.001* (.001)	.001 (.001)	.001 (.001)
MidCI[a]		-.242* (.103)	-.335** (.124)	-.317** (.109)	-.286** (.108)	-.285** (.108)	-.293** (.108)	-.304** (.108)
LateCI[b]		-.452*** (.121)	-.655*** (.147)	-.577*** (.130)	-.586*** (.128)	-.555*** (.129)	-.558*** (.129)	-.594*** (.129)
MidCI*Time			.012 (.009)	.012 (.009)	.012 (.009)	.012 (.009)	.009 (.009)	.010 (.009)
LateCI*Time			.027* (.011)	.026* (.011)	.026* (.011)	.026* (.011)	.018 (.011)	.020 (.011)
Mastery				-.321*** (.019)	-.317*** (.019)	-.317*** (.019)	-.308*** (.018)	-.423*** (.057)
Educ- <HS[c]					.345* (.156)	.311* (.156)	.206 (.154)	.199 (.153)
Educ- Some HS [d]					.244* (.099)	.221* (.099)	.174 (.097)	.177 (.096)
Educ- HS Grad[e]					.104 (.069)	.106 (.069)	.090 (.068)	.084 (.067)
Educ – Some College[f]					.064 (.069)	.051 (.069)	.042 (.068)	.038 (.067)
Married[g]					-.146* (.058)	-.134* (.058)	-.126* (.057)	-.127* (.057)
Income					-1.81^{-6} (1.44^{-6})	-1.56^{-6} ($1.43^{-6)}$	-1.29^{-6} (1.4^{-6})	-1.33^{-6} (1.39^{-6})
Male[h]						-.014 (.052)	.007 (.051)	.008 (.050)
Black[i]						.193* (.075)	.166* (.073)	.167* (.073)
OthRace[j]						.211 (.154)	.206 (.150)	.209 (.149)
Function							-.302*** (.034)	-.300*** (.034)
Comorbid							.039 (.023)	.043** (.023)
Mastery*MidCI								.115 (.060)
Mastery*LateCI								.190** (.073)

Random Effects

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Intercept	.582*** (.054)	.559*** (.052)	0.558*** (.052)	.422*** (.043)	.403*** (.041)	.402*** (.041)	.403*** (.041)	.402*** (.041)
Intercept By Time	-.049*** (.010)	-.047*** (.010)	-.047*** (.010)	-.047*** (.009)	-.046*** (.009)	-.046*** (.009)	-.045*** (.009)	-.046*** (.009)
Time	.016*** (.003)	.016*** (.003)	.016*** (.003)	.016*** (.003)	.016*** (.003)	.016*** (.003)	.016*** (.003)	.016*** (.003)
Residual	.296*** (.014)	.296*** (.014)	.295*** (.014)	.275*** (.013)	.275*** (.013)	.274*** (.013)	.266*** (.012)	.266*** (.012)

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
-2LL	5416.4	5402.2	5395.8	5131.0	5105.0	5096.9	5006.9	5000.1
AIC	5436.4	5426.2	5423.8	5161.0	5147.0	5144.9	5058.9	5056.1
AICC	5436.5	5426.3	5424.0	5161.2	5147.4	5145.5	5059.6	5056.9
BIC	5479.2	5477.6	5483.8	5225.3	5237.0	5247.8	5170.4	5176.2
Chi Square	713.98***	704.45***	707.19***	487.82***	462.01***	454.42***	460.93***	449.52***
ΔD	---	14.2**	6.4*	264.8**	26.0**	8.1*	90.0**	6.8*

***p<.001 ** p< .01 * p<.05

Note: raw estimates are presented first along with standard errors in parentheses.

[a-b] reference category is: early onset chronic illness. [c-f] reference categories are: college graduate. [g] reference category is: unmarried. [h] reference category is: female. [i-j] reference categories are: white.

In model 3, I included interactions of time with timing of onset dummy variables, "mid-life onset" and "late-life onset" and concluded that no statistically significant difference in rate of change in depressive symptoms exists between adults with early versus mid-life onset (b=.012). However, a significant difference in rate of change was detected between adults with early versus late-life onset (b=.027, p<.05). Late-life onset is associated with .027 units faster change in depressive symptoms than early onset. The relationship between timing of illness onset and depressive symptoms is graphically illustrated below in figure 5.4, which depicts that prior to illness onset occurring (baseline), young adults report higher depressive symptoms than middle age or older adults. All three age groups experience an initial decrease in depressive symptoms that levels off during the latter half of the study period (≈year 11) and begins a gradual increase towards the end of the 16 year study period.

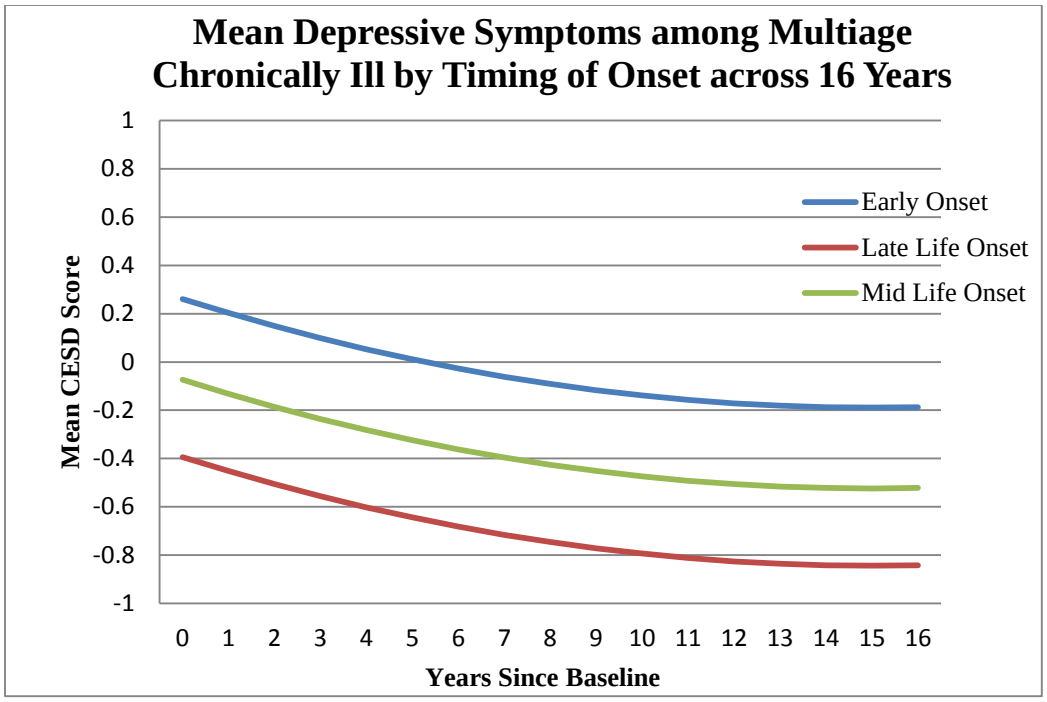

Figure 5.4 Mean Depressive Symptoms among Multiage Chronically Ill by Timing of Onset across 16 Years (Table 5.4, Model 3)

Even after including timing of illness onset's interaction with time, the main effects of mid-life and late-life onset remained significant at the .01 and .001 levels respectively. Hypothesis 4a stated that early onset chronic illness is associated with greater depressive symptoms than mid-life or late-life onset. This hypothesis was supported because adults with early onset illness consistently report higher levels of depressive symptoms than peers with later onset, even when mastery is controlled (model 4).

In model 4, I tested hypothesis 4b, which states that mastery mediates the relationship between timing of illness onset and depressive symptoms. Mean mastery was a significant predictor of depressive symptoms ($b=-.321$, $p<.001$), reiterating its protective function as a coping resource. Higher mean mastery was associated with lower depressive symptoms. After entering mastery into the model, estimates of differences between late-life and early onset and mid-life and early onset remained statistically significant, but decreased in effect size. The effect size of late-life onset on depressive symptoms decreased to -.577 ($p<.001$), meaning that when

controlling for mean mastery, older adults' depressive symptoms become more similar to adults with early onset illness. However, as demonstrated in the examination of the timing of chronic illness-mastery relationship in chapter 4, this bivariate relationship was not statistically significant.

Similarly, mastery's inclusion in model 4, decreased the effect size for mid-life onset (b=-.317, p<.01). Although a significant difference remains, depressive symptoms for adults with mid-life and early onset become slightly more similar once mean mastery is included in the model. The reduction in effect sizes for both mid-life and late-life onset demonstrates that mastery has an effect on the effects of timing of illness onset on depressive symptoms. However, its function as a mediator was not supported. Consequently, hypothesis 4b was not supported.

Model 5 includes socioeconomic status (SES) characteristics as control variables. Educational attainment lower than high school completion was significantly and positively associated with depressive symptoms (b=.345, p<.05; b=.244, p<.05). Being married was negatively associated with depressive symptoms (b=-.146, p<.05). Annual income was not a significant predictor of depressive symptoms. Controlling for SES characteristics, main effects of mid-life onset and late-life onset remained significant at the .01 and .001 levels respectively. However, effect size for mid-life onset decreased noticeably from -.317 to -.286, suggesting that SES explains some of the difference in mean mastery between those with early versus mid-life illness onset. Late-life onset increased in effect size from -.577 to -.586 and retained significance. As in previous models, there was no significant difference in rate of change in depressive symptoms between adults with early and mid-life onset. Yet, depressive symptoms for adults with late-life onset continue to change at a rate that is .026 units faster than adults with early onset illness. Mastery remained significant, but decreased in effect size (b=.317, p<.001).

I added additional demographic characteristics to model 6. Sex and being a "non-white/non-black" person were not significant predictors of depressive symptom. Black adults with chronic illness reported depressive symptoms that were .193 units higher than white and "other race" adults. The inclusion of sex and race into the model did not change significance of mid-life onset, late-life onset, late-life*time, mastery, education, or marital status from the previous model. However, one noteworthy change was the decrease in effect size for all of these variables, with the exception of late-life onset's interaction with time.

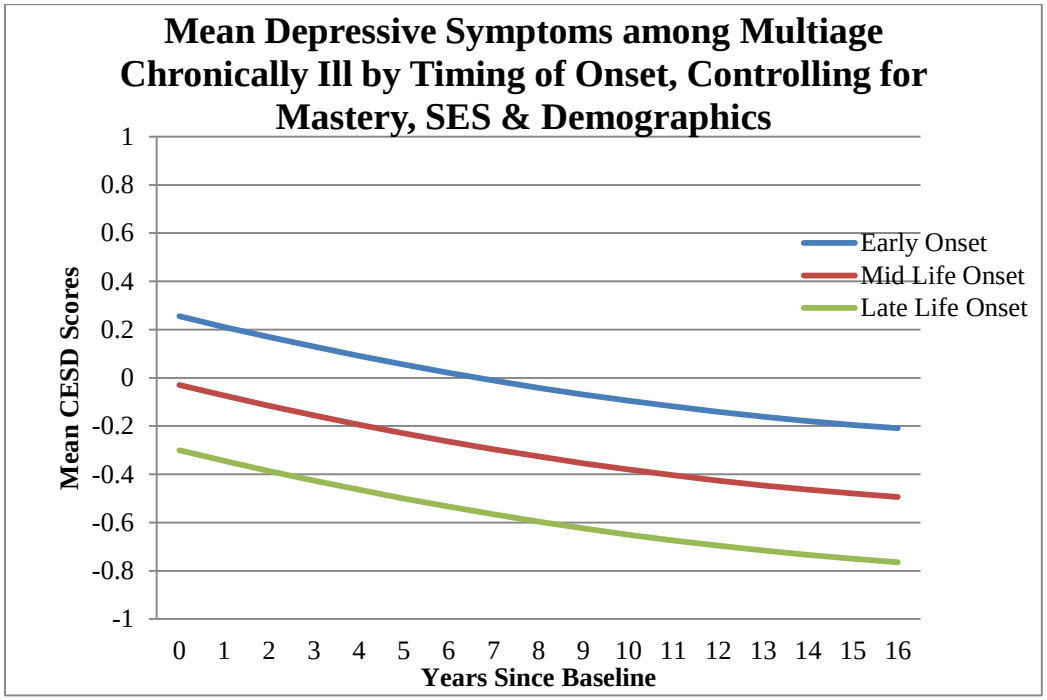

Figure 5.5 Mean Depressive Symptoms among Multiage Chronically Ill by Timing of Onset, Controlling for Mastery, SES, and Demographics (Table 5.4, Model 6)

Figure 5.5 above illustrates this relationship between timing of illness onset and depressive symptoms, controlling for mastery, SES, and demographic characteristics. Controlling for the effects of these predictors, the curves appear more steep, suggesting more rapid change in standard CESD scores. Depressive symptoms do not appear to level off until after the study period has concluded. However, the ordering of the age groups remains consistent with prior

models' findings that adults with early onset illness report higher depressive symptoms than those with mid-life onset who report higher depressive symptoms than adults with late-life onset.

In model 7, I included illness characteristics as control variables. Mean functional health was a significant predictor of depressive symptoms (b= -.302, p<.001), with better functioning being associated with lower depressive symptoms. Including functional impairment into the model increased the effect size for mid-life onset from -.285 to -.293 and late-life onset from -.555 to -.558. The number of comorbid chronic conditions was not a significant predictor of depressive symptoms.

A statistically significant difference in mean depressive symptoms between adults with early onset as compared to adults with mid-life (b=-.293, p<.01) and adults with late-life (b=-.558, p<.001) remained after controlling for illness characteristics. However, controlling for these characteristics resulted in there being no difference in rate of change by timing of illness onset. Education also became a non-significant predictor. Mastery remained highly significant (p<.001) and slightly decreased in effect size from -.317 to -.308. Marital status and black race remained significant predictors of depressive symptoms.

Lastly, hypothesis 4c was tested in model 8. To review, hypothesis 4c stated that mastery moderates the relationship between timing of illness onset and depressive symptoms. I included interactions between mastery and mid-life onset and mastery and late-life onset into the model. The interaction between mastery and late-life was statistically significant (p<.01), meaning that mastery's effect differs for adults with early onset as compared to those with late-life onset. Specifically, adults with late-life onset experience a protective effect from mastery that is .190 units larger than adults who experience illness onset prior to 36 years of age. The main effects of

mid-life and late-life onset, mastery, marital status, black race, and functional impairment remained significant.

5.4.2 Random Effects

In table 5.4, estimates for random effects inform us that considerable heterogeneity exists in baseline depressive symptoms and changes in depressive symptoms across the 16 year study period. In all models, random intercept and random slope are negatively associated ($p<.001$), which means that subjects with higher baseline depressive symptoms tend to have slower growth, represented by less steep slopes. For example, in model 1, for every one unit increase on the CESD scale at baseline, a person experiences changes in depressive symptoms at a rate that is .049 slower than a person with unit lower on the CESD. Estimates of the residual across models 1-6 affirm that considerable variance in depressive symptoms (22.4% - 36.8%) remains unexplained and is attributable to unobservable within-person factors.

5.4.3 Summary

In review, results presented in table 5.2 confirm the following:

1. Young adults report significantly higher levels of depressive symptoms than do older adults or middle age adults, even when controlling for mastery, demographic characteristics, and illness characteristics.

2. Mean mastery does not explain group differences in growth of depressive symptoms for adults with mid-life onset and early onset.

3. The protective function provided by mastery depends on when in life illness onset occurs. Thus, mastery moderates the effect of timing of illness onset on depressive symptoms for adults with early versus late-life onset. Specifically, older adults benefit from greater protection against depressive symptoms from the mastery they possess than do young adults with early onset.

4. Heterogeneity in baseline and growth of depressive symptoms exists between and within people.

5. Individual variation in depressive symptoms prior to illness onset (e.g. baseline) partially explains interindividual and intraindividual variation in growth of depressive symptoms.

In the chapter that follows, I discuss findings presented in chapter 4 and in this chapter in relation to the cumulative disadvantage and youthful resilience explanations previously presented and conclude with directions for future research.

6 CONCLUSION

In this concluding chapter, I will reassert the aims of this project and describe how I have achieved each through analyses presented in chapters 4 and 5. I will discuss the hypotheses tested and findings in relation to the opposing explanatory frameworks previously discussed, cumulative disadvantage and youthful resilience. Lastly, I will revisit the strengths and limitations of this work for consideration in future studies relating to chronic illness onset.

6.1 Aims

In review, the primary aims of this project were 1) to apply a life course perspective to the examination of the timing of chronic illness onset, 2) to situate "ill-timed" chronic illness within the stress process model as a primary stressor that generates conditions favorable to secondary stressors, and 3) to explore the process through which mastery mediates and/or moderates the psychological effects of chronic illness onset. Through analyses presented in chapters 4 and 5 of this document, I have accomplished each of these aims. Below, I review the hypotheses and major findings of this study and subsequently, discuss them in relationship to the cumulative disadvantage and youthful resilience explanations.

6.2 Review of Hypotheses & Findings

In this section, I review each of the hypotheses tested and summarize related findings immediately below each hypothesis.

Hypothesis 1a. Early onset chronic illness is significantly associated with lower mastery compared to those without early onset chronic illness.

- Among this sample, early onset chronic illness is not associated with lower mean mastery than remaining healthy, even when controlling for socioeconomic and demographic characteristics.

Hypothesis 2a. Early onset chronic illness is significantly associated with higher depressive symptoms compared to those without early onset chronic illness.

- Adults who later develop early onset chronic illness report higher levels of depressive symptoms prior to illness onset than do peers who remain healthy.

- There is no statistical difference in the rate of change in depressive symptoms among adults with early onset illness and healthy peers.

Hypothesis 2b. Mastery mediates the relationship between early onset chronic illness and depressive symptoms.

- Mastery does not mediate the effect of early onset chronic illness on depressive symptoms.

Hypothesis 3a. Early onset chronic illness is associated with lower mastery than illness onset at mid-life or late-life.

- Mid-life onset and late-life onset are not significantly associated with higher mastery than onset during young adulthood (24-35), even when controlling for socioeconomic and demographic characteristics.

- Timing of illness onset is not predictive of rate of change in mastery across the 16 year study period.

Hypothesis 4a. Early onset chronic illness is associated with greater depressive symptoms than illness onset at mid-life or late-life.

- Among chronically ill adults, young adults report significantly higher levels of depressive symptoms than do older adults or middle age adults, even when controlling for mastery, demographic characteristics, and illness characteristics.

Hypothesis 4b. Mastery mediates the relationship between timing of illness onset and depressive symptoms.

- Mean does not mediate or explain group differences in growth of depressive symptoms for adults with mid-life onset and early onset.

Hypothesis 4c. Mastery moderates the relationship between timing of illness onset and depressive symptoms.

- The protective function provided by mastery depends on when in life illness onset occurs. Thus, mastery moderates the effect of timing of illness onset on depressive symptoms for adults with early versus late-life onset.

In the section that follows, I discuss how each of these findings support or do not support the

cumulative disadvantage and youthful resilience explanations presented throughout this work.

6.3 Cumulative Disadvantage versus Youthful Resilience

The cumulative disadvantage explanation posited that the onset of chronic illness later in the life course would be less harmful to the individual because mastery accrues over time. Thus, middle age and older adults who become ill during these stages would be relatively advantaged in the acquisition of this protective resource when compared to people who become ill early as young adults. In conjunction with a life course perspective (Elder 1994) and stress process (Pearlin et al. 1989), the timing of this disruptive life event and stressor differentially positions some chronically ill people to experience more secondary stressors, like increased depressive symptoms, than others.

The findings that mid-life and late-life onset were not significantly associated with higher mastery than early onset did not support the cumulative disadvantage explanation. This finding likely reflects that the samples in this study were overrepresented by ill adults with higher than average mastery. Within each age group of chronically ill adults, there was less variation in mastery scores than would be found in the general population. Due to patterns of attrition, the late-life group was also significantly overrepresented by women and white adults. Each of these demographic characteristics were associated with higher mean mastery (Mirowsky and Ross 2003).

In considering the cumulative disadvantage explanation, the findings that among the young adult sample and all ages restricted to chronically ill sample, baseline mastery is highly predictive of rate of change in mastery are particularly interesting. These findings do support a cumulative disadvantage explanation because a person's level of mastery at baseline (prior to illness onset), contributes to how quickly they acquire and/or lose mastery across the 16 year period. Irrespective of age at illness onset, those who are advantaged in this coping resource less

mastery at baseline. Within the stress process framework (Pearlin et al. 1981), this finding illustrates how contextual and historical factors, such as demographics and prior life experiences, differentially expose people to stressors and their widely varying effects (Pearlin and Schooler 1978, Schieman et al. 2003).

Support for a cumulative disadvantage explanation is much stronger within the findings on the effects of chronic illness onset on depressive symptoms. Early onset chronic illness is associated with higher depressive symptoms than illness onset at mid-life or late-life, even when controlling for mastery, demographics, and illness characteristics. In this study, I argued that although illness onset is a personally disruptive experience at any age, it is the individual and social expectations of youth that make early onset particularly harmful (Hobbie et al. 2000). Chronic illness earlier than expected may contribute to higher depressive symptoms than later onset because the socially defined developmental stage and the lived experience of illness are incongruent (Comeaux and Jaser 2010, Saunders et al. 2011). The relative advantage experienced by adults with mid-life and late-life onset is occupying developmental stages that are more congruent with their socially defined status as chronically ill (Ornstein et al. 2013, Paez et al. 2009).

Findings that mastery moderates the effects of timing of illness onset on depressive symptoms provide additional support for the cumulative disadvantage explanation. Mastery provides a larger benefit or buffering effect against depressive symptoms (Shanahan and Bauer 2004) for chronically ill adults with late-life onset than for adults with early onset illness. Consequently, becoming ill as an older adult is less harmful, as the mastery that has been acquired is more effectively mobilized when the stressor of illness occurs. Conversely, onset during young adulthood is a relative disadvantage because young adults report the highest levels

of depressive symptoms but garner less protection against depressive symptoms from the mastery they possess.

As discussed above, findings mostly supported the cumulative disadvantage explanation. However, some findings provide limited support for the alternative explanation I termed youthful resilience. Youthful resilience referred to the idea that the disruptive experience of chronic illness onset is less harmful to the young because self-concepts are relatively more malleable than in later stages (Easterbrooks et al. 2013, Karatsoreos and McEwen 2011, Wiebe et al. 2005). Thus, illness onset and the identity as chronically ill should be more readily integrated into the young adult's sense of self (Stiles 2000). Support for youthful resilience is limited, however, findings that baseline levels of mastery and rate of change in mastery do not differ between adults with early, mid-life, and late-life onset support that young adults are not disadvantaged in this capacity. In analyses that compared mastery trajectories of chronically ill young adults and healthy/ never ill young adults, the ill fared as well as healthy peers, providing some support for a youthful resilience explanation. These findings suggest that among these young people, health status is not predictive of mastery. It is also possible that differences among this age cohort do not begin to appear until later in the life course beyond the 16 year window of this study. In the section below, I review the strengths and limitations of this work.

6.4 **Strengths**

Throughout this project, I have repeatedly asserted that current scholarship has consistently overlooked the importance of timing in examining chronic illness as a lived experience. Most often this work (Gignac et al. 2000; Lyons et al. 2009) has presented the experiences of middle age and older adults as wholly representative of chronically ill people without explicit recognition of young adults for whom illness onset is socially non-normative

(Fuligni and Pederson 2002, Saunders et al. 2011). I have sought to address this inattention to early onset illness through this dissertation project; as I believe that this scholarly misrepresentation partially contributes to the inaccurate social perception of chronic illness as a lived experience of the aged.

In their seminal work, Neugarten et al. (1965) suggested that "there is a prescriptive timetable for the ordering of major life events" and that "expectations regarding age-appropriate behavior form an elaborated and pervasive system of norms governing behavior and interaction, a network of expectations that is imbedded throughout the cultural fabric of adult life" (pg. 711). Chronic illness onset at a socially non-normative time frays "the cultural fabric of adult life" by challenging presumptions about the normative life course. Moreover, early or "ill-timed" chronic illness serves as an example of a less common, yet nonetheless, important area of study in life course studies. In this project, I have situated timing as a central feature of illness and consequently, offered an alternative perspective on young adult psychosocial development and the diversity within the chronic illness experience.

To summarize, the strengths of this work are 1) the project's focused attention on the chronic illness-mastery association among adults with early onset illness, 2) the examination of chronic illness as a stressor that contributes to depressive symptoms, 3) the within group comparison of age cohort differences in changes in coping resources and mental health among the chronically ill, and 4) analyses of longitudinal data in examining the effects of the enduring stressor of chronic illness. As a result, this project contributes to the collective understanding of chronic illness as a personal life event and social phenomenon.

6.5 Limitations

The primary limitation of this study is the impact of across wave attrition on the study sample. Thorough discussion of attrition, missing data, and considerations needed in interpretation of results has been presented in chapter 3. It is worth noting again that this study's samples and the findings derived from them represent the best case scenario for chronic illness' impact on the coping resource, mastery. Due to the across wave attrition of men, black adults, and those with the lowest levels of education, there is an overrepresentation of women, white adults, and those with at least a high school education. As the demographic characteristics of those most likely to attrit are also characteristics most predictive of low mastery (Mirowsky and Ross 2003), the sample is skewed toward adults with the highest levels of psychological coping.

Additional limitations of the present study have been identified and are discussed below. First, the chronically ill samples include young adults who became ill at different points of the study and may have a wide range of duration of illness. The experience of a 27 year old who reports illness at time 1 may differ from someone who becomes ill at the cusp of mid-life at 34 years old at time 3. Secondly, diagnoses represented among the chronically ill range in severity and type. Due to the reliance on pre-constructed ACLS measures of health status, health conditions include multiple forms of arthritis, Types 1 and 2 of diabetes, various cancers etcetera. Analyses do not control for illness differences as immediately life threatening, mild and asymptomatic, etcetera. Lastly, the measure of depressive symptoms is but one measure of depression and may reflect biases inherent to the CESD tool. Although other measures of depressive symptoms (i.e. CIDI) were included in American Changing Lives Study data, the CESD has been used widely in social science research with nationally representative samples (Levine 2013).

Additionally, the American Changing Lives Study data used in this study was first collected in 1986, nearly 30 years prior to the completion of the present study. The datedness of the data must be considered when interpreting findings. Specifically, the social meanings of the age groups/ life stages at the center of this study (young adults, mid-life, late life) are likely linked to the period in which the data was collected. My conceptualization and operationalization of these three life stages may differ from how respondents themselves and society at large viewed these life stages in 1986.

Similarly, findings that suggest differences by timing of illness onset may reflect cohort effects that are inherent to the historical contexts in which each group passed through each life stage (e.g. young adults in 1986 experienced childhood in late 1950s through 1960's as compared to older adults in 1986 experienced childhood in late 1890s to early 1920s). In conjunction with a life course perspective, these potential cohort differences may explain differences in depressive symptoms, conceptualization of one's self as chronically ill, and the significance of chronic illness as a life event at a particular life stage.

6.6 Directions for Future Research

The present study has made contributions to the sociological study of chronic illness by 1) applying the life course principal, timing in lives to the onset of chronic illness, 2) examining chronic illness onset within the stress process as a stressor defined by the context of age, and 3) establishing that protective coping resources, specifically, mastery, have an inequitable effect across subgroups experiencing the same stressor, chronic illness onset. Completion of this project has also highlighted multiple directions for future research pertaining to chronic illness onset as a significant life event. In the section below, I present a few areas in need of further study or consideration.

One of the primary challenges in conducting the present study was the limited data that included chronically ill people under the age of 40 as a significant proportion of a study's sample. The American Changing Lives Study was selected for this study because it did include longitudinal data for people who become ill as young adults, as I defined them in this study (24-35 year old). However, this data set still had limitations. The ACLS did not include data on the youngest young adults, those between 18 and 23, what is typically considered "college age." Future data collection in large scale, nationally representative surveys focused on physical and mental health should include ample cases from this age demographic. This data would be particularly valuable to other scholars interested in the transition from adolescence into adulthood and the enduring consequences of disruptive life events that occur during this distinct period of the life course.

Also, although across wave attrition and the analytic challenges that it creates are common when analyzing secondary panel data, sampling adjustments (e.g. extensive oversampling of men with less than high school completion) that ensure demographic representativeness would be beneficial. The questions that I have asked in this study interrogated the relationship between physical health (the body), mental health (the mind), and self-concept (the self). Few nationally representative quantitative data sets solicit information that can be used to answer these types of questions. Although there is great value to inferential statistics, future work that seeks to examine these types of multilayered questions would be augmented by a mixed methods approach.

Additionally, at the core of this project is the acknowledgement that even among people facing the same stressor, context is important in defining the stressor and its long term consequences. Due to this project's foundation in a life course perspective, I have highlighted

age as the primary context in which illness onset occurs. However, it is reasonable to assume that other individual contexts like race, gender, and socioeconomic status, also differentially position people for better or worse outcomes when faced with the stressor of chronic illness. These areas require further study and should be considered in future work in this arena.

Lastly, additional research is needed in examining how other known protective resources, namely self-esteem (Simoni 2006) and social support (Pearlin 1981), influence the timing of illness onset – mastery and timing of illness onset- depressive symptoms relationships. Although work in the area of chronic illness has been done with consideration of self-esteem and social support, none have applied a life course perspective and considered how age at onset contextualizes the experience and its effects.

As a significant life event, stressor, and "biographical disruption" (Bury 1982) to nearly half of American adults, chronic illness and the effects of its timing of onset are ripe for sociological study. Through this project, I have contributed to this work by acknowledging and examining the lived experience of a previously understudied population, chronically ill young adults, and its unique experience with chronic illness.

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7 APPENDICES

Appendix A: Results Derived from Multiple Imputation

Table 7.1 Model Fit Statistics on Imputed Data (Mastery as Outcome among Young Adult Sample)

(N=2188)	Model 1	Model 2	Model 3	Model 4
	mastery	mastery, time (fixed)	mastery, time (fixed & random)	mastery, time (fixed & random), time_sq (fixed & random)
AIC	5935.1	5931.8	5821.0	5734.3
AICC	5935.1	5931.9	5821.0	5734.4
BIC	5948.0	5949.1	5846.8	5777.3
-2LL	5929.1	5923.8	5809.0	5714.3
Chi Square	410.55***	412.42***	527.26***	607.67***
ΔD[22]	---	5.3*	117.8**	94.7**

***p<.001 ** p< .01 * p<.05

[22] ΔD denotes the change in deviance from one model to the subsequent model. The values presented represent the change in -2LL and its significance when compared to the X^2 value that corresponds with the difference in degrees of freedom from a model to the next model.

Table 7.2 2 Level MLM (Imputed) - Mastery (Young Adult Sample) (N=2188)

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	.029 (.036)	.035 (.037)	.041 (.094)	-.064 (.010)	-.295 (.180)
Time	.036*** (.010)	.036*** (.010)	.027* (.013)	.036*** (.010)	.036*** (.010)
Time Squared	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)
Yngci[a]		-.048 (.082)	-.014 (.010)	-.013 (.080)	-.011 (.080)
Yngci*time			.010 (.010)	---	---
Educ- <HS[b]				-.452 (.230)	-.435 (.230)
Educ- Some HS [c]				-.091 (.109)	-.082 (.109)
Educ- HS Grad[d]				-.094 (.074)	-.082 (.075)
Educ – Some College[e]				.087 (.073)	.096 (.073)
Married[f]				-.092 (.060)	.096 (.060)
Income				6.42^{-6}*** (1.69^{-6})	6.6^{-6}*** (1.69^{-6})
Male[g]					-.079 (.054)
Black[h]					.037 (.068)
Othrace[i]					.156 (.116)

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5
-2LL	5929.1	5713.9	5712.8	5680.9	5676.4
AIC	5935.1	5735.9	5736.8	5714.9	5716.4
AICC	5935.1	5736.0	5736.9	5715.2	5716.8
BIC	5948.0	5783.3	5788.4	5788.1	5802.5
Chi Square	410.55***	607.79***	606.97***	594.76***	587.67***
ΔD	---	215.2**	1.1	31.9**	4.5

***p<.001 ** p< .01 * p<.05

Note: raw estimates are presented first along with standard errors in parentheses.

[a] reference category is: healthy. [b-e] reference categories are: college graduate. [f] reference category is: unmarried. [g] reference category is: female. [h-i] reference categories are: white.

Table 7.3 Model Fit Statistics on Imputed Data (Mastery as Outcome among the All Ages Restricted to Chronically Ill Sample) (N=3120)

(N=3120)	Model 1	Model 2	Model 3	Model 4
	mastery	mastery, time (fixed)	mastery, time (fixed & random)	mastery, time (fixed & random), time_sq (fixed & random)
AIC	8935.2	8888.1	8738.4	8598.1
AICC	8935.2	8888.1	8738.4	8618.1
BIC	8949.2	8906.7	8766.3	8618.2
-2LL	8929.2	8880.1	8726.4	8664.7
Chi Square	556.43***	573.53***	727.24***	824.57***
ΔD	---	49.1**	153.7**	61.7**

***p<.001 ** p< .01 * p<.05

Table 7.4 Level MLM on Mastery among the All Ages Restricted to Chronically Ill Sample (N=3120)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept	.050 (.034)	.031 (.038)	.018 (.039)	-.059 (.083)	-.088 (.085)	-.399** (.144)
Time	.038*** (.010)	.038*** (.001)	.040*** (.009)	.038*** (.010)	.038*** (.009)	.042*** (.010)
Time_Sq	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)	-.003*** (.001)
MidCI[a]		-.012 (.087)	-.013 (.108)	.043 (.085)	.027 (.085)	.015 (.084)
LateCI [b]		.090 (.006)	.156* (.076)	.172** (.060)	.159** (.060)	.173** (.060)
MidCI*Time			.003 (.010)	---	---	---
LateCI*Time			-.010 (.007)	---	---	---
Educ- <HS[c]				-.355** (.112)	-.378*** (.112)	-.324** (.112)
Educ- Some HS[d]				-.171 (.094)	-.180 (.093)	-.161 (.093)
Educ- HS Grad[e]				-.108 (.070)	-.102 (.070)	-.094 (.069)
Educ – Some College[f]				.009 (.072)	.003 (.071)	.010 (.071)
Married[g]				-.054 (.055)	-.071 (.055)	-.073 (.055)
Income				6.046^{-6}*** (1.411^{-6})	5.72^{-6}*** (1.405^{-6})	5.63^{-6}*** (1.40^{-6})
Male[h]					.142** (.050)	.134** (.050)
Black [i]					.029 (.066)	.035 (.066)
OthRace[j]					-.295* (.147)	-.287* (.146)
Function						.079** (.029)
Comorbid						-.017 (.022)

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
-2LL	8598.1	8595.8	8593.6	8542.1	8529.3	8520.4
AIC	8618.1	8619.8	8621.6	8578.1	8571.3	8566.4
AICC	8618.2	8619.9	8621.8	8578.4	8571.6	8566.8
BIC	8664.7	8675.7	8686.9	8662.0	8669.1	8673.6
Chi Square	824.57***	822.51***	824.16***	791.68***	759.92***	755.95***
ΔD	---	2.3	2.2	51.5**	12.8**	8.9*

***p<.001 ** p< .01 * p<.05

Note: raw estimates are presented first along with standard errors in parentheses.

[a-b] reference category is: early onset chronic illness. [c-f] reference categories are: college graduate. [g] reference category is: unmarried. [h] reference category is: female. [i-j] reference categories are: white.

Table 7.5 Model Fit Statistics on Imputed Data (CESD as Outcome among Young Adult Sample) (N=2188)

(N=2188)	Model 1	Model 2	Model 3	Model 4
	CESD	CESD, time (fixed)	CESD, time (fixed & random)	CESD, time (fixed & random), time_sq (fixed & random)
AIC	5894.8	5826.5	5798.2	5761.9
AICC	5894.8	5826.5	5798.2	5762.0
BIC	5907.7	5843.7	5824.0	5805.0
-2LL	5888.8	5818.5	5786.2	5741.9
Chi Square	533.49***	563.69***	595.99***	630.18***
ΔD	---	70.3**	32.3**	44.3**

***p<.001 ** p< .01 * p<.05

Table 7.6 2 Level MLM- CESD (Young Adult Sample) (N=2188)

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	.091* (.041)	.060 (.043)	.056 (.043)	.063 (.038)	.226*** (.085)
Time	-.059*** (.010)	-.059*** (.043)	-.058*** (.010)	-.047*** (.010)	-.045*** (.010)
Time Squared	.002*** (.001)	.002*** (.001)	.002*** (.001)	.002** (.001)	.001** (.001)
Yngci[a]		.236** (.091)	.269* (.108)	.219** (.077)	.154* (.073)
Yngci*time			-.005 (.008)	---	---
Mastery				-.346*** (.020)	-.344*** (.020)
Educ- <HS[b]					.003 (.208)
Educ- Some HS[c]					.160 (.098)
Educ- HS Grad[d]					.076 (.067)
Educ – Some College[e]					.017 (.066)
Married[f]					-.162** (.055)
Income					-5.19^{-6}*** (1.52^{-6})
Male[g]					-.051 (.049)
Black[h]					.289*** (.063)
Othrace[i]					.242* (.104)

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5
-2LL	5741.9	5735.2	5734.9	5470.8	5388.7
AIC	5761.9	5757.2	5758.9	5494.8	5430.7
AICC	5762.0	5757.4	5759.1	5494.9	5431.2
BIC	5805.0	5804.6	5810.6	5546.4	5521.1
Chi Square	630.18***	622.28***	622.47***	350.46***	311.99***
ΔD	---	6.7**	0.3	264.1**	82.1**

***p<.001 ** p< .01 * p<.05

Note: raw estimates are presented first along with standard errors in parentheses .

[a] reference category is: healthy. [b-e] reference categories are: college graduate. [f] reference category is: unmarried. [g] reference category is: female. [h-i] reference categories are: white.

Table 7.7 Model Fit Statistics on Imputed Data among the All Ages Restricted to Chronically Ill Sample (N=3120)

(N=3120)	Model 1	Model 2	Model 3	Model 4
	CESD	CESD, time (fixed)	CESD, time (fixed & random)	CESD, time (fixed & random), time_sq (fixed & random)
AIC	8317.7	8306.5	8203.1	8104.9
AICC	8317.7	8306.5	8203.1	8105.0
BIC	8331.7	8325.1	8231.1	8151.5
-2LL	8311.7	8298.5	8191.1	8084.9
Chi Square	886.79***	892.84***	100.17***	1097.80***
ΔD	---	13.2**	107.4**	106.2**

***p<.001 ** p< .01 * p<.05

Table 7.8 2 Level MLM on CESD among the All Ages Restricted to Chronically Ill Sample (N=3120)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Intercept	-.067* (.034)	-0.060 (.039)	-.038 (.040)	-.037 (.036)	.020 (.074)	-.036 (.076)	.0989*** (.121)	1.002 (.121)
Time	-.032*** (.010)	-.032*** (.010)	-.035** (.008)	-.028*** (.008)	-.022** (.008)	-.022** (.008)	-.032*** (.008)	-.031*** (.008)
Time_Sq	.002** (.001)	.002*** (.001)	.002** (.001)	.001* (.001)	.001 (.001)	.001 (.001)	.001 (.001)	.001 (.001)
MidCI[a]		.246* (.090)	.344** (.108)	.351*** (.095)	.301* (.094)	.288** (.094)	.295* (.094)	.223** (.073)
LateCI[b]		-.132*** (.062)	-.288*** (.075)	-.252*** (.067)	-.309*** (.067)	-.268*** (.067)	-.029*** (.067)	-.189*** (.052)
MidCI*Time			-.014 (.008)	-.014 (.008)	-.014 (.008)	-.014 (.008)	-.009 (.008)	.223** (.073)
LateCI*Time			.022*** (.006)	.020*** (.067)	.019*** (.006)	.019*** (.006)	.014* (.006)	-.189*** (.052)
Mastery				-.321*** (.015)	-.320*** (.015)	-.321*** (.015)	-.310*** (.015)	-.324*** (.018)
Educ- <HS[c]					.421*** (.099)	.379*** (.099)	.268** (.097)	.272*** (.097)
Educ- Some HS[d]					.235** (.083)	.211* (.082)	.144 (.081)	.143** (.080)
Educ- HS Grad[e]					.142* (.062)	.130* (.061)	.103 (.060)	.097 (.060)
Educ – Some College[f]					.083 (.063)	.065 (.063)	.045 (.062)	.043 (.061)
Married[g]					-.141** (.048)	-.121* (.048)	.111* (.047)	-110* (.047)
Income					-2.48^{-6} (1.25^{-6})	-2.06^{-6} (1.24^{-6})	-1.78^{-6} (1.21^{-6})	-1.8^{-6} (1.21^{-6})
Male [h]						-.026 (.044)	.003 (.043)	.003 (.043)
Black[i]						.230*** (.057)	.214*** (.057)	.215*** (.056)
OthRace[j]						.230 (.129)	.226 (.127)	.227 (.126)
Function							-.261*** (.024)	-.268 (.024)*
Comorbid							.053** (.018)	.058 (.018)
Mastery*MidCI								-.043 (.051)
Mastery*LateCI								.077 (.036)*

Model Fit

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
-2LL	8084.9	8039.7	8052.5	7651.1	7592.8	7574.4	7436.1	7439.2
AIC	8104.9	8067.7	8080.5	7681.1	7634.8	7622.4	7488.1	7491.2
AICC	8105.0	8067.8	8080.6	7681.2	7635.1	7622.8	7488.6	7491.7
BIC	8151.5	8132.9	8145.7	7751.0	7732.6	7734.2	7609.3	7612.4
Chi Square	1097.80***	1058.42***	1096.34***	783.73***	734.94***	726.80***	750.64***	731.40***
ΔD	---	45.2**	|12.8|**	401.0**	58.3**	18.4**	138.3**	|3.1|

***p<.001 ** p< .01 * p<.05

Note: raw estimates are presented first along with standard errors in parentheses.
[a-b] reference category is: early onset chronic illness. [c-f] reference categories are: college graduate. [g] reference category is: unmarried. [h] reference category is: female. [i-j] reference categories are: white.